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ATLAS OF

METHODS OF CLINICAL INVESTIGATION,

WITH AN EPITOME OF

CLINICAL DIAGNOSIS

AND OF

SPECIAL PATHOLOGY AND TREATMENT

OF

INTERNAL DISEASES.

BY

DR. CHRISTFRIED JAKOB,
Formerly First Assistant in the Medical Clinic at Erlangen.

AUTHORIZED TRANSLATION FROM THE GERMAN.

EDITED BY

AUGUSTUS A. ESHNER, M.D.,
Professor of Clinical Medicine in the Philadelphia Polyclinic; Physician to the Philadelphia Hospital, etc.

With 182 Colored Illustrations upon 68 Plates, and 64 Illustrations in the Text.

PHILADELPHIA:
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EDITOR'S NOTE.

Of books on clinical medicine there is no deficiency, but the beauty, the clearness, and the accuracy of the illustrations in the original German volume, of which the present is a translation, seemed to be sufficient justification for adapting the work to the needs of American medical students and practitioners. As is indicated in the preface, a good illustration will often convey more and leave a deeper impression than the most elaborate description.

Extensive modifications in the text have not been found necessary, and the translation has been free rather than literal, the endeavor having been to convey the spirit rather than the language of the original. The German volume has attained a deservedly large measure of popularity, and it is hoped that this reproduction in English will prove equally acceptable and useful.

The original has been followed in placing trichinosis among infectious diseases of undetermined origin, although its dependence upon the trichina spiralis is recognized, and the parasite itself is described in its proper place.

It has been thought well to add an index.

65649

PREFACE.

THIS Atlas of the methods of clinical investigation contains a selection of the most important clinical conditions a knowledge of which the recent practitioner may acquire more readily and retain more permanently through good illustrations than through the most elaborate description.

The various methods of investigation have been delineated as uniformly as possible. The illustrations of Part I are all from original preparations;[1] those of Part II are derived mostly from patients under immediate observation.

For the text of the Epitome, supplementary to the plates, the principle seemed sufficient that a portion of our diagnostic knowledge could with advantage be drawn from a concisely arranged compendium; never, however, therapeutic modes of procedure; for the latter the more elaborate text-book will be required.

CHR. JAKOB.

BAMBERG, January, 1897.

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[1] The drawings were made under my direction by Herr Krapf.

DESCRIPTION OF PLATES.

PART I.

CLINICAL MICROSCOPY AND CHEMIC COLOR-REACTIONS.

PLATE 1.—The Red Blood-corpuscles (Erythrocytes).

Fig. 1. Fresh preparation of normal blood.
Fig. 2. Stained preparation of normal blood.
Fig. 3. Abnormal variations in size of the red blood-corpuscles.
Fig. 4. Abnormal variations in form of the red blood-corpuscles.
Fig. 5. Normal frogs' blood, stained.
Fig. 6. Nucleated red blood-corpuscles of man.

PLATE 2.—White Blood-corpuscles (Leukocytes).

Fig. 1. Polynuclear leukocytes.
Fig. 2. Mononuclear leukocytes.
Fig. 3. Eosinophile cells (α-granules).
Fig. 4. Basophile and neutrophile granulation.
Fig. 5. Medullary cells (splenomedullary leukemia).
Fig. 6. Medullary cells, with nuclei in process of division.

PLATE 3.—The Blood in Various Diseases.

Fig. 1. Simple profound anemia attending chronic nephritis.
Fig. 2. Pernicious anemia.
Fig. 3. Hemoglobinemia.
Fig. 4. Leukocytosis attending croupous pneumonia (fresh).
Fig. 5. Leukocytosis attending pneumonia (stained).
Fig. 6. Blood-preparation from a case of typhoid fever.

PLATE 4.—The Blood-state of Leukemia.

Fig. 1. Fresh blood-preparation from a subject dead of leukemia.
Figs. 2, 3, 4. Stained preparations from a case of splenomedullary leukemia.
Fig. 5. Lymphatic leukemia.
Fig. 6. Acute leukemia in a child half a year old.

PLATE 5.—Parasites of the Blood.

Fig. 1. Blood from a subject dead of anthrax.
Fig. 2. Blood from a case of pyemia.
Fig. 3. Pneumococci.
Fig. 4. Spirilla of relapsing fever.
Fig. 5. Blood from a case of malaria.
Fig. 6. Malarial plasmodia.

PLATE 6.—Blood-spectra and Blood-crystals.

Fig. 1. Normal solar spectrum, oxyhemoglobin-spectrum, spectrum of reduced hemoglobin, of methemoglobin, of reduced CO-hemoglobin, of hematin.
Fig. 2. Hematoidin-crystals.
Fig. 3. Teichmann's hemin-crystals.

PLATE 7.—Microscopy of the Mouth and the Nasal Cavities.

Fig. 1. Gingival deposit.
Fig. 2. Thrush-fungus.
Fig. 3. Spirochetæ from a case of gangrenous stomatitis.
Fig. 4. Secretion from the nasal mucous membrane.
Fig. 5. Diphtheria-bacilli.
Fig. 6. Section through diphtheria-membrane.

PLATE 8.—Microscopy of the Sputum.

Fig. 1. Normal sputum (unstained).
Fig. 2. Normal sputum (stained).
Fig. 3. Pigment- (soot) cells.
Fig. 4. Pigment- (cardiac-lesion) cells.
Fig. 5. Sputum from an attack of asthma (unstained).
Fig. 6. Sputum from a case of asthma (stained).

PLATE 9.

Fig. 1. Sputum from an abscess of the lung.
Fig. 2. Sputum from gangrene of the lung.
Fig. 3. Fibrinous coagulum.

PLATE 10.—The Parasites of the Sputum.

Fig. 1. Pneumococci in pneumonic sputum.
Fig. 2. Staphylococci from an abscess of the lung.
Figs. 3, 4. Sputum from a case of tuberculosis.
Fig. 5. Sputum from a case of influenza-bronchitis.
Fig. 6. Sputum from a case of actinomycosis of the lung.

PLATE 11.—Microscopy of Contents of Stomach and Intestines.

Fig. 1. General view of gastric contents.
Fig. 2. General view of the feces.
Fig. 3. Intestinal contents from a case of cholera nostras.
Fig. 4. Intestinal contents from a case of cholera asiatica.

PLATE 12.—The Most Important Color-reactions of the Gastric Juice.

Litmus-paper, congo-paper, phloroglucin-vanillin test, methyl-violet reaction, Uffelmann's reagent.

PLATE 13.—Urinary Sediments.

Fig. 1. Brickdust sediment.
Fig. 2. Yellowish friable sediment.
Fig. 3. Bloody sediment.
Fig. 4. Sodium urate.
Fig. 5. Uric-acid crystals.

PLATE 14.—CRYSTALLINE URINARY SEDIMENTS.

Fig. 1. Calcium oxalate.
Fig. 2. Hippuric acid.
Fig. 3. Ammonio-magnesium phosphate, triple phosphate.
Fig. 4. Ammonium urate.
Fig. 5. Calcium carbonate.
Fig. 6. Calcium sulphate and calcium phosphate.

PLATE 15.—CRYSTALLINE URINARY SEDIMENTS.

Fig. 1. Basic magnesium phosphate.
Fig. 2. Cystin.
Fig. 3. Leucin.
Fig. 4. Tyrosin.
Fig. 5. Cholesterin.
Fig. 6. Earthy phosphates and bacteria.

PLATE 16.—ORGANIZED URINARY SEDIMENTS.

Fig. 1. Squamous epithelium from urethra and bladder.
Fig. 2. Epithelial cells from the pelvis of the kidney.
Fig. 3. Renal epithelium.
Fig. 4. Pus-cells.
Fig. 5. Red blood-corpuscles.
Fig. 6. Spermatozoa.

PLATE 17.—URINARY TUBE-CASTS.

Fig. 1. Cylindroids and hyaline tube-casts.
Fig. 2. Coarsely and finely granular tube-casts.
Fig. 3. Waxy tube-casts.
Fig. 4. Blood-casts.
Fig. 5. Fatty casts.

PLATE 18.—URINARY SEDIMENTS ATTENDING DISEASES OF THE BLADDER AND THE KIDNEYS.

Fig. 1. Sediment from cystitis.
Fig. 2. Sediment from calculous pyelitis.
Figs. 3, 4. Sediment from acute hemorrhagic nephritis.
Fig. 5. Chronic nephritis—large red, variegated kidney.
Fig. 6. Chronic nephritis—large white kidney.

PLATE 19.

Fig. 1. Sediment from chronic parenchymatous nephritis.
Fig. 2. Sediment from true contracted kidney.
Fig. 3. Sediment from febrile albuminuria.
Fig. 4. Sediment from amyloid kidney.
Fig. 5. Pus from gonorrhea.
Fig. 6. Pus from tuberculous cystitis.

PLATE 20.—THE MOST IMPORTANT COLOR-REACTIONS OF THE URINE.

Figs. 1, 2, 3. Trommer's test for sugar.
Fig. 4. Bismuth-test.
Fig. 5. Moore's (caramel-) test.
Fig. 6. Ferric-chlorid reaction in diabetes.

Fig. 7. Peptone-test.
Fig. 8. Indican-test.
Fig. 9. Test for biliary coloring-matter.
Fig. 10. Heller's blood-test.
Fig. 11. Test for melanin.
Fig. 12. Diazo-reaction.

PLATE 21.—Demonstration of some Medicaments in the Urine.

Fig. 1. Demonstration of antipyrin.
Fig. 2. Demonstration of salicylic acid.
Fig. 3. Demonstration of bromin (potassium bromid).
Fig. 4. Demonstration of iodin (potassium iodid).
Contents of cysts, abscesses, etc.
Fig. 5. Crystals of uric acid from a gouty nodule.
Fig. 6. Hematoidin-crystals from an old abscess.
Fig. 7. Echinococcus-fluid (evaporated).
Fig. 8. Echinococcus-fluid (sediment).

PLATE 22.—The Most Important Pyogenic Micro-organisms.

Fig. 1. Staphylococcus pyogenes albus.
Fig. 2. Streptococcus pyogenes.
Fig. 3. Bacterium coli commune.
Fig. 4. Proteus vulgaris.
Fig. 5. Bacillus of glanders.
Fig. 6. Typhoid-bacilli.

PART II.

NORMAL PROJECTION OF THE VISCERA AND PERCUTORY TOPOGRAPHY.

PLATE 23.—Projection of the Internal Organs upon the Anterior Aspect of the Body.

PLATE 24.—Percutory and Auscultatory Topography.

PLATE 25.—Projection of the Internal Organs upon the Posterior Aspect of the Body.

PLATE 26.—Percutory Topography of the Back.

PLATE 27.—Projection of the Internal Organs upon the Lateral Aspects of the Trunk.

PLATE 28.—Percutory Topography of the Lateral Aspects of the Trunk.

SCHEMATA OF DISEASES OF LUNGS AND HEART.

PLATE 29.—Croupous Pneumonia of the Posterior Inferior Portion of the Left Side.

PLATE 30.—Croupous Pneumonia.

PLATE 31.—Exudative Pleurisy on the Left Side Posteriorly.

PLATE 32.—Exudative Pleurisy on the Left Side.

PLATE 33.—Exudative Pleurisy on the Right Side.
PLATE 34.—Hydrothorax.
PLATE 35.—Acute Diffuse Bronchitis.
PLATE 36.—Bronchopneumonia.
PLATE 37.—Pulmonary Infarction.
PLATE 38.—Pulmonary Emphysema.
PLATE 39.—Incipient Pulmonary Tuberculosis.
PLATE 40.—Progressive Pulmonary Tuberculosis.
PLATE 41.—Pneumothorax.
PLATE 42.—Pyopneumothorax.
PLATE 43.—Uncompensated Cardiac Lesion. HEART.
PLATE 44.—Compensated Mitral Stenosis.
PLATE 45.—Mitral Insufficiency.
PLATE 46.—Aortic Insufficiency.
PLATE 47.—Tricuspid Insufficiency.
PLATE 48.—Idiopathic Hypertrophy of the Heart.
PLATE 49.—Exudative Pericarditis.
PLATE 50.—Aneurism of the Aorta.

DIAGRAMMATIC REPRESENTATION OF ABDOMINAL DISEASES.

PLATE 51.

Fig. *a*. Carcinoma of the cardia, with stenosis of the esophagus.
Fig. *b*. Normal and pathologic positions of the stomach.

PLATE 52.—Cicatricial Stenosis of the Pylorus.
PLATE 53.—Dilatation of the Stomach, with Carcinoma of the Pylorus.
PLATE 54.—Carcinoma of the Stomach.
PLATE 55.—Constricted Liver.
PLATE 56.—Cirrhosis of the Liver.
PLATE 57.—Melanosarcoma of the Liver.
PLATE 58.—Cholelithiasis; Hydrops of the Gall-bladder.
PLATE 59.—Amyloid Degeneration.
PLATE 60.—Splenomedullary Leukemia.
PLATE 61.—Pancreatic Cyst.
PLATE 62.—Ascites attending Cardiac Lesion.
PLATE 63.—Carcinoma of the Intestine.
PLATE 64.—Occlusion of the Bowel; Invagination.
PLATE 65.—Perityphlitis.
PLATE 66.—Tuberculous Peritonitis.
PLATE 67.—Perforative Peritonitis.
PLATE 68.—Sarcoma of the Right Kidney.

CONTENTS.

SECTION I.

PAGE
EXAMINATION OF THE PATIENT 1–18
    History 1–6
    Present State 6–16
    Subjective Symptoms 16–18

SECTION II.

GENERAL CONSIDERATIONS UPON METHODS OF EXAMINATION . 19–34
    Microscopy 20–22
    Examination with Mirrors 22
    Thermometry 23
    Exploratory Puncture 23
    Bacteriologic Methods 24–28
    Palpation 28
    Percussion 29–31
    Auscultation 31–34

SECTION III.

SPECIAL DIAGNOSIS OF DISEASES OF THE INTERNAL ORGANS . 35–130
    I. Examination of the Respiratory Apparatus 35–54
        1. Nose and Nasopharyngeal Space 35
        2. Larynx and Trachea 36–40
        3. Lungs 40–48
            Anatomic 40
            Physiologic 41
            Inspection 42
            Spirometry 42
            Percussion 42–45
            Auscultation 46–48
        4. Puncture of the Pleura 49
        5. The Sputum 50–54
    II. Examination of the Circulatory Apparatus 54–79
        1. Heart 54–66
            Anatomic 54
            Physiologic 54
            Theory of Valvular Lesions 55–60
            Derangement of Compensation 60
            Inspection and Palpation 61
            Percussion 62–64
            Auscultation 64–66

PAGE
2. The Blood-vessels 66–72
The Pulse 67–72
3. The Blood 72–79
III. Examination of the Digestive Apparatus 79–99
1. Mouth and Pharynx 79–81
2. Esophagus 81
3. Stomach 82–89
4. Liver 90–92
5. Spleen 92
6. Abdomen, Intestines, and Peritoneum 93–99
The Stools 97–99
IV. Examination of the Uropoietic System 99-115
1. Kidneys 99
2. Bladder 100
3. The Urine 100–115
V. Examination of Abnormalities of Metabolism 115–119
VI. The Most Important Parasites 120–130

SECTION IV.

EPITOME OF SPECIAL PATHOLOGY AND TREATMENT 131–232
I. Infectious Diseases 131–161
A. Bacterial Infections 131–144
1. Tuberculous Diseases 131–137
2. Typhoid Fever 137–140
3. Diphtheria and Croup 140
4. Influenza 141
5. Asiatic Cholera 142
6. Leprosy 143
7. Anthrax 143
8. Glanders 143
B. Coccus-infections 144–150
9. The Rheumatic Diseases 144–146
Acute Rheumatic Polyarthritis 144
Acute Endocarditis 145
Acute Rheumatic Pleurisy 146
10. Cryptogenetic Septicopyemia 146
11. Croupous Pneumonia 147–149
12. Epidemic Cerebrospinal Meningitis 149
13. Erysipelas 150
C. Actinomyces-infections 150
D. Spirillum-infections 151
14. Relapsing Fever 151
E. Plasmodial Infections 151–153
15. Malarial (intermittent) Fever 151–153
F. Infectious Diseases of Undetermined Origin 153–161
The Acute Exanthemata 153
16. Morbilli (measles) 154
17. Scarlet Fever 155
18. Rubeola (Rötheln) 155
19. Variola (smallpox) 155-157
20. Varicella (chickenpox) 157
21. Epidemic Parotiditis (mumps) 157
22. Pertussis (whooping-cough) 157

PAGE
23. Cholera Morbus (cholera nostras) 158
24. Dysentery 158
25. Typhus Fever (spotted fever) 159
26. Hydrophobia (canine rabies) 160
27. The Plague 160
28. Yellow Fever 160
29. Trichinosis 160
II. Diseases of the Respiratory Organs 161–177
A. Diseases of the Nose 161–162
1. Acute and Chronic Rhinitis 161
2. Ozena 162
3. Epistaxis 162
B. Diseases of Larynx, Trachea, and Bronchi 163–167
4. Acute and Chronic Laryngitis 163
5. Edema of the Larynx 164
6. Tuberculosis of the Larynx 164
7. Syphilis of the Larynx 164
8. Paralysis of the Laryngeal Muscles (see p. 38) 164
9. Spasm of the Glottis (laryngismus stridulus) 164
10. Tumors of the Larynx 164
11. Acute and Chronic Bronchitis 165
12. Fetid Bronchitis 166
13. Bronchiectasis 166
14. Bronchial Asthma 167
C. Diseases of Lungs and Pleura 167–177
15. Croupous Pneumonia (see p. 147) 167
Differential Diagnosis between Pneumonia and Pleurisy 167
16. Bronchopneumonia 168
17. Atelectasis of the Lung 168
18. Pulmonary Emphysema 169
19. Pulmonary Edema 170
20. Pulmonary Tuberculosis (see p. 132) 170
21. Pulmonary Gangrene 170
22. Pulmonary Abscess 171
23. Pneumonokonioses 171
24. Pulmonary Embolism (hemorrhagic infarction) 171
25. Pleurisy 172–175
26. Pneumothorax 175
27. Hydrothorax 176
28. New-growths in the Chest 176
III. Diseases of the Circulatory Organs 177–190
A. Diseases of the Heart 177–186
1. Acute Endocarditis 177
Table of Valvular Lesions 178–179
2. Valvular Lesions of the Heart 180–184
3. Idiopathic Hypertrophy of the Heart 184
4. Diseases of the Myocardium 185
5. Neurotic Disorders of the Heart 186
B. Diseases of the Pericardium 186–189
6. Pericarditis 186–188
7. Hydropericardium, Hemopericardium, Pneumopericardium 188–189
C. Diseases of the Bloodvessels 189

PAGE
8. Arteriosclerosis 189
9. Aneurism of the Aorta 190
IV. Diseases of the Digestive Organs 190-211
A. Diseases of the Mouth and the Pharynx 190
1. Stomatitis 190
2. Glossitis 192
3. Angina (tonsillitis) 192
4. Chronic Hypertrophy of the Tonsils 193
5. Dry Chronic Pharyngitis 193
B. Diseases of the Esophagus 194
6. Stenosis of the Esophagus 194
C. Diseases of the Stomach 195-199
7. Acute Gastritis 195
8. Chronic Gastritis 195
9. Gastric Ulcer 196
10. Carcinoma of the Stomach 197
11. Dilatation of the Stomach 198
Pyloric Stenosis, Table 199
12. Nervous Dyspepsia 199
D. Diseases of the Intestines 199-202
13. Intestinal Catarrh 199
14. Intestinal Tuberculosis 200
15. Carcinoma of the Bowel 201
16. Stenosis and Occlusion of the Bowel (ileus) 201
17. Habitual Constipation 202
18. Intestinal Parasites 202
E. Diseases of the Peritoneum 202-205
19. Perityphlitis 202
20. Acute Peritonitis 203
21. Chronic Peritonitis 204
22. Ascites 204
23. Carcinoma of the Peritoneum 205
F. Diseases of the Liver 205-210
24. Catarrhal Jaundice 205
25. Cholelithiasis 206
26. Cirrhosis of the Liver 207
27. Acute Yellow Atrophy of the Liver 207
28. Carcinoma of the Liver 208
29. Syphilis of the Liver 208
30. Abscess of the Liver 208
31. Echinococcus of the Liver 208
32. Cyanotic (nutmeg) Liver 209
Fatty Degeneration of the Liver 209
Amyloid Liver 209
33. Constricted Liver 209
34. Thrombosis and Inflammation of the Portal Vein . . 209
Table of Diseases of the Liver 210
G. Diseases of Spleen 210
35. Enlargement of the Spleen 210
H. Diseases of the Pancreas 211
V. Diseases of the Uropoietic Organs 211-220
A. Diseases of the Kidneys 211-218
1. Acute Hemorrhagic Nephritis 211
2. Chronic Nephritis 212

PAGE
3. True Contracted Kidney 213
4. Cyanotic Kidney 214
Hemorrhagic Infarction 215
5. Amyloid Kidney 215
Table of Diseases of the Kidney 215
6. Pyelonephritis 215
7. Nephrolithiasis 216
8. Tuberculosis of the Kidney 217
9. Tumors of the Kidney (see p. 99) 217
10. Hydronephrosis 217
11. Wandering Kidney 218
B. Diseases of the Bladder 218-220
12. Cystitis 218
13. Tumors of the Bladder 219
14. Nocturnal Enuresis 219
VI. Diseases of the Blood and of the Bodily Metabolism . 220-227
A. Diseases of the Blood 220-224
1. Chlorosis 220
2. Anemia 220
3. Leukemia 221
4. Hemoglobinemia 223
5. Hemorrhagic Diathesis 223
B. Disorders of the Bodily Metabolism 224-227
6. Diabetes Mellitus, Diabetes Insipidus 224
7. Uric-acid Diathesis (gout) 225
8. Obesity 226
VII. Diseases of the Joints and Bones 227-228
1. Rachitis 227
2. Osteomalacia 227
3. Deforming Arthritis 227
4. Muscular Rheumatism 228
VIII. Some Important Forms of Poisoning 228-232

SECTION V.

THERAPEUTIC NOTES 233-242
I. Dietetic Methods of Treatment 233-234
1. Dietary for Diseases of the Stomach 233
2. Fever-diet 233
3. Rest-cure 233
4. Reduction-cure (for obesity) 234
5. Nutrient Enemata 234
6. Artificial Foods 234
II. Hydrotherapy 234-236
III. Treatment by Climate and Baths 236
IV. Physical Methods of Treatment 236-237
V. The Most Important Medicaments 237-242

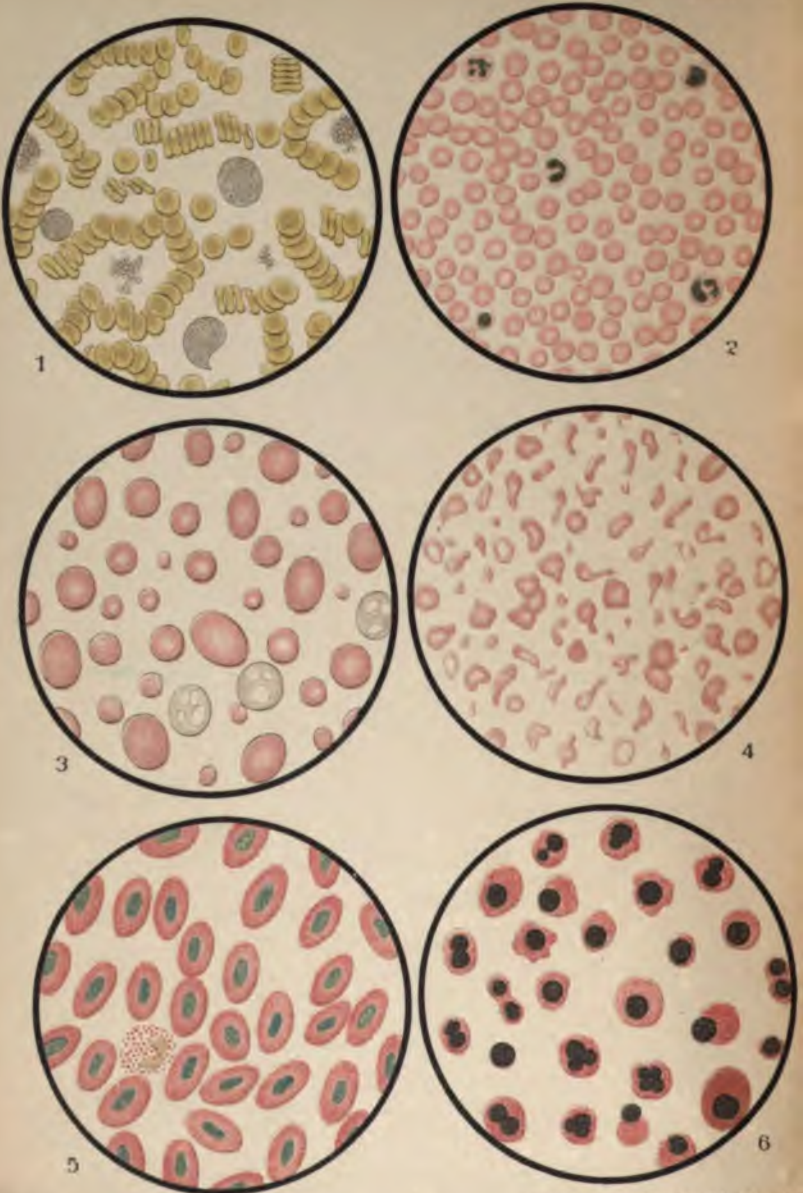
CLINICAL MICROSCOPY

AND

CHEMIC COLOR-REACTIONS.

(PLATES 1-22.)

Tab. 1.



Lith. Anst F. Reichhold, Münche

PLATE 1.

THE RED BLOOD-CORPUSCLES (ERYTHROCYTES).

FIG. 1. **Fresh Preparation of Normal Blood.**—The red blood-corpuscles mostly in rouleaux; some with distinct central depressions; others standing on edge (biscuit-shaped); all of approximately the same size (7.6 μ). Three white blood-corpuscles with distinct granulation of the protoplasm; that in the middle coarsely granular; the others finely granular. Several groups of blood-plates (like clusters of grapes).—Magnified 350 times.

FIG. 2. **Stained Preparation of Normal Blood.**—The red blood-corpuscles deeply stained with eosin; in the center (corresponding with the concavity) less deeply. Five leukocytes with readily recognizable nuclei (three polynuclear, two mononuclear. See Plate 2).—Magnified 350 times. Stained with hematoxylin and eosin.

Note.—In this, as in all subsequent illustrations of the blood, the relation between the white and the red blood-corpuscles will be correct if the field as portrayed be conceived to contain three times as many red blood-corpuscles as white, the number of white remaining as in the illustration.

FIG. 3. **Abnormal Variations in Size of the Red Blood-corpuscles.**—Showing abnormally small (microcytes, from 2.2 μ downward), abnormally large red corpuscles (macrocytes, up to 15 μ). Some of the macrocytes are stained partially or totally of a violet hue instead of the normal red tint of eosin (anemic degeneration, alteration in tingibility of the stroma). Others present lighter areas (hemoglobinemic degeneration).—Magnified 400 times. Stained with methylene-blue and eosin.

FIG. 4. **Abnormal Variations in Form of the Red Blood-corpuscles.**—The red blood-corpuscles show all possible variations of form, excrescences, etc. This condition is designated poikilocytosis.—Magnified 350 times. Stained with eosin.

FIG. 5. **Normal Frog's Blood, Stained.**—The red blood-corpuscles are much larger than in man, of oval shape, and provided with longitudinal nuclei. A white blood-corpuscle with eosinophile granulations (see Plate 2).—Magnified 300 times. Stained with methylene-blue and eosin.

FIG. 6. **Nucleated Red Blood-corpuscles of Man.**—The red blood-corpuscles of man are normally non-nucleated (only in the fetal state are they nucleated). In some diseases (leukemia, anemia) nucleated red corpuscles also appear, and in the form of erythrocytes of normal size (normoblasts) or as macrocytes = [illegible] (gigantoblasts). The nuclei may be in part absorbed; in part, however, they show intense staining of the chromatin, undergo division (by budding), and may escape free from the red blood-corpuscles (free nuclei). The significance of this process (regeneration [?], transformation into white blood-corpuscles, especially mononuclear [?]) is yet a matter of doubt.

= Crenation in normal blood (if seen [illegible];
2 hrs. after mixing get necrobiotic ch. [illegible]

PLATE 2.

WHITE BLOOD-CORPUSCLES (LEUKOCYTES).

FIG. 1. **Polynuclear Leukocytes.** gr 4 —They show partly lobulated and fissured, partly completely separated, nuclei. In the degenerated (dying) forms the nuclei are stained more feebly and the protoplasm undergoes disintegration (karyolysis).—Magnified 700 times. Stained with methylene-blue and eosin (*upper half*), hematoxylin and eosin (*lower half*).

FIG. 2. **Mononuclear Leukocytes.**—They are distinctly smaller and display only a narrow zone of protoplasm. A distinction is made between a large and a small form. Magnification and staining the same as in Fig. 1. LYMPHOCYTES.

FIG. 3. **Eosinophile Cells** (*a*-**Granules**).—Leukocytes filled with bright bodies, unstained presenting a coarsely granular appearance, and staining intensely with eosin; they contain the *a*-granules (Ehrlich). Eosinophile cells are mostly polynuclear; strong pressure may cause them to rupture, with escape of the granules. A distinction is made between large, medium-sized, and small forms. Normally they exhibit ameboid movement. In addition to their presence in the blood (from 2 or 5 to 15%) they are found also in the sputum (especially in cases of asthma), in nasal mucus, in gonorrheal pus, etc. The significance of the granules has not yet been determined.—Magnified 700 times. Stained with methylene-blue and eosin. If the stained preparation is rapidly washed in 10% potassic hydrate solution, the *a*-granules appear with especial distinctness. gr 1.

FIG. 4. **Basophile and Neutrophile Granulation.**—*Left upper quadrant:* Leukocytes with (mast-cell) granulation (*γ*-granules). The polynuclear cells contain moderately coarse granules that stain deeply with methylene-blue, dahlia, etc. *Right upper quadrant:* Leukocytes with fine basophile granulation (*δ*-granules), especially in the marginal zone (mononuclear forms). *Lower half:* Leukocytes with neutrophile (violet) granulation (*ε*-granules). They correspond with the majority of the polynuclear leukocytes (and are found, also, in sputum and in pus in large numbers) and contain a fine, dust-like granulation. *Left:* stained with eosin; *right:* with Ehrlich's triacid solution. —Magnified 800 times. [illegible]

FIG. 5. **Medullary Cells (Spleno-medullary Leukemia).** —These cells are ordinarily not found in normal blood, but appear in large numbers in some varieties of leukemia. They have invariably large lobulated nuclei that usually do not stain deeply and generally exhibit neutrophile granulation. Some contain, also, esinophile granules. They are derived from the bone-marrow and, in contrast with the remaining leukocytes, display no ameboid movement.

FIG. 6. **Medullary Cells, with Nuclei in Process of Division.**—From a case of leukemia. There were found the various stages of indirect karyokinesis (aster, constriction, skein-form). —*Magnified* 1000 times. Stained with hematoxylin.

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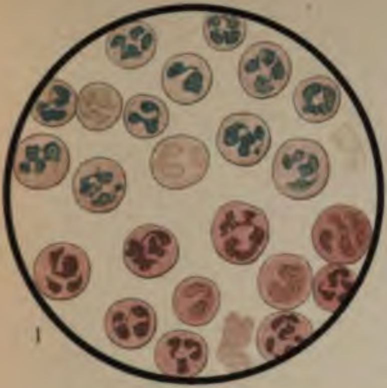

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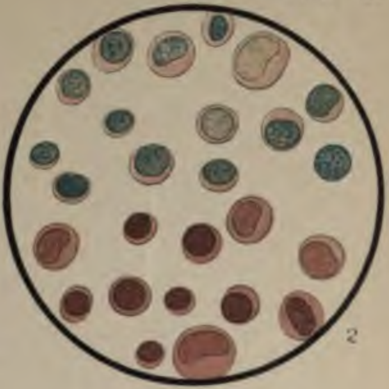

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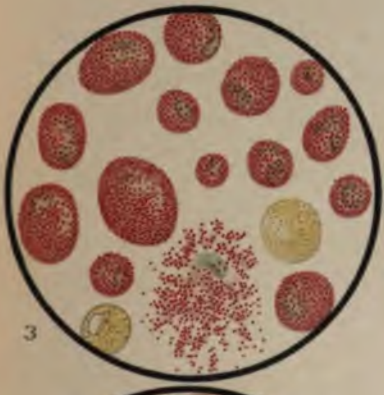

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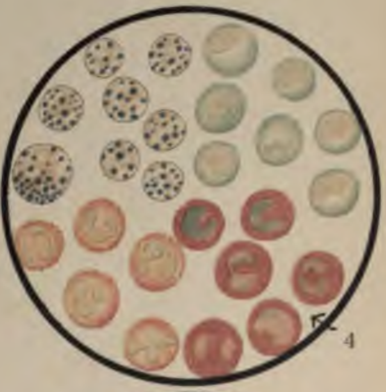

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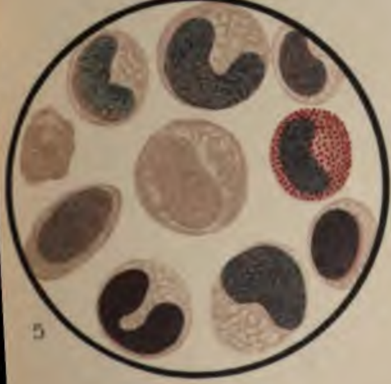

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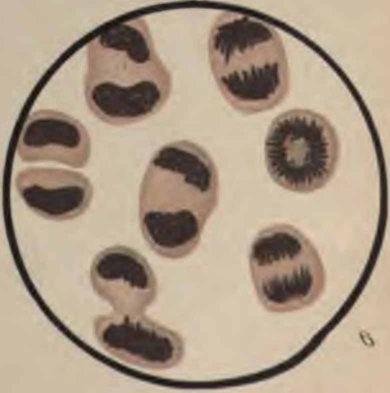

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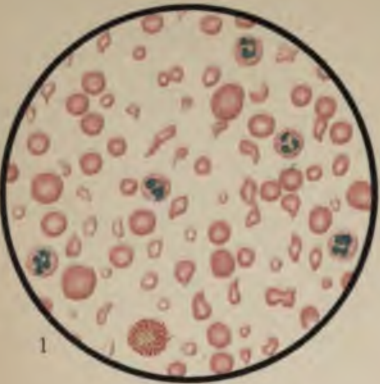
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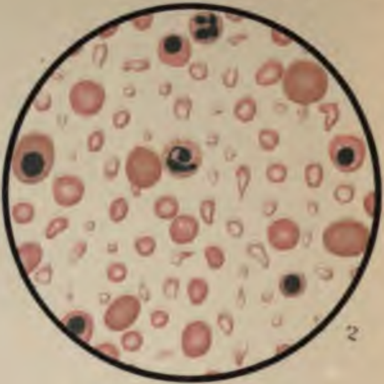
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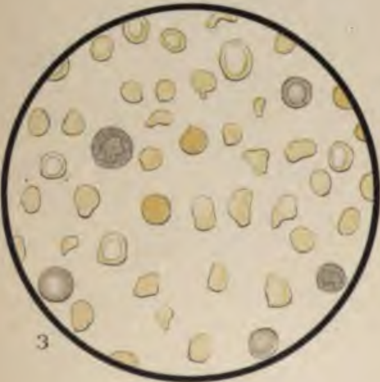
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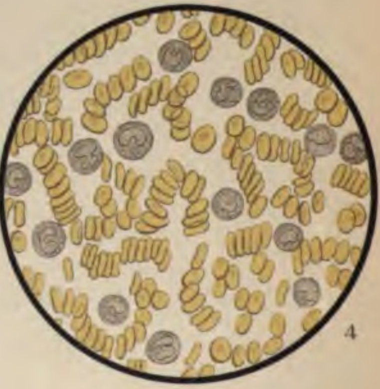
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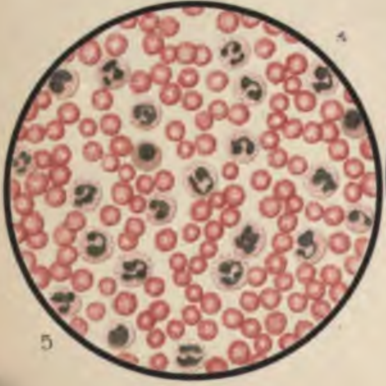
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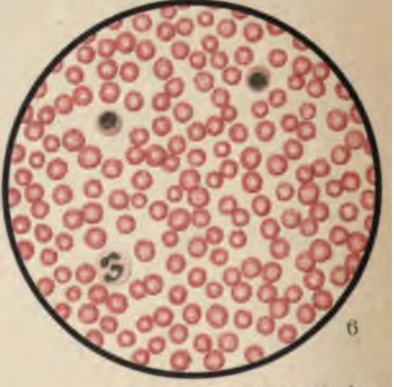
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PLATE 3.

THE BLOOD IN VARIOUS DISEASES.

FIG. 1. **Simple Profound Anemia** attending chronic nephritis. The red corpuscles exhibit poikilocytosis, and some microcytes and macrocytes are present. Nucleated red corpuscles are wanting. The white corpuscles are somewhat increased (leukocytosis) and are mostly polynuclear, with one eosinophile cell. The enumeration showed 1,700,000 red and 18,000 white corpuscles to the cubic millimeter, with only 42% of hemoglobin.—Magnified 300 times. Stained with hematoxylin and eosin.

FIG. 2. **Pernicious Anemia.**—Marked poikilocytosis. Many microcytes and macrocytes, the latter showing anemic degeneration. Fairly numerous nucleated red corpuscles (normoblasts and gigantoblasts). White corpuscles somewhat increased. Enumeration: 600,000 red, 12,000 white corpuscles; hemoglobin, 30%. Staining and magnification the same as in Fig. 1.

FIG. 3. **Hemoglobinemia** (fresh preparation).—In a case of poisoning with potassic chlorate (prescribed as a gargle) there were found in the fresh blood numerous red corpuscles which had lost their hemoglobin totally or partially, and were swollen, pale, and changed in form (shadows and poikilocytes). The spectroscopic examination showed the methemoglobin-line in the red (see Plate 6, Fig. 1). The blood was of brownish hue.

FIG. 4. **Leukocytosis** attending croupous pneumonia (fresh preparation).—During the course of a number of febrile diseases (pneumonia, cerebro-spinal meningitis, scarlet fever, etc.) there occurs throughout the course of the attack marked increase in the polynuclear leukocytes in the blood of the capillaries (unequal distribution ?). If on examination of a fresh preparation of such blood an average of more than from 5 to 8 leukocytes is found in a single field, leukocytosis of about 15,000 may be presumed to exist; when the number of white corpuscles is from 12 to 15, the leukocytosis is more than 20,000. In the preparation depicted there were more than 56,000 white blood-corpuscles to the cubic millimeter (determined by counting).

FIG. 5. **Leukocytosis** attending pneumonia (stained with hematoxylin and eosin).—The increase involves exclusively the polynuclear forms; no eosinophile cells are present.

FIG. 6. **Blood-preparation from a Case of Typhoid Fever.**—In contrast with pneumonia there is here no leukocytosis, but rather a diminution of the polynuclear elements. Staining the same as in Fig. 5.

PLATE 4.

BLOOD-STATE OF LEUKEMIA.

FIG. 1. **Fresh Blood-preparation from a Subject dead of Leukemia.**—The white blood-corpuscles are enormously increased. A count during life showed 360,000 white blood-corpuscles and 2,600,000 red blood-corpuscles to the cubic millimeter. Finely and coarsely granular (*a*-granules) leukocytes are easily recognized; on the addition of acetic acid the nuclei also become distinct; there are many mononuclear cells (especially large forms). In the blood after death Charcot-Leyden crystals have formed in large numbers (they are not to be seen during life).

FIG. 2. **Stained Preparation from a Case of Splenomedullary Leukemia.**—Stained with methylene-blue and eosin.—Magnified 300 times.

FIG. 3. Preparation from the same blood stained with hematoxylin and eosin.

FIG. 4. Preparation of the same blood stained with triacid solution.

In Figs. 2 and 3 the red corpuscles are stained red, the nuclei blue, the *a*-granules red, the basophile granules blue; in Fig. 4 the red corpuscles are stained yellow, their nuclei green, the nuclei of the leukocytes bluish, the *a*-granules red, the neutrophile granules violet.

All of the preparations exhibit moderate poikilocytosis; the white blood-corpuscles are markedly increased. This increase is contributed to by the polynuclear neutrophile cells, and especially the eosinophile cells; besides there are present (in contrast to leukocytosis) large mononuclear medullary cells, in part exhibiting *a*-granulation. There are many nucleated red blood-corpuscles.

FIG. 5. **Lymphatic Leukemia**—Stained with hematoxylin and eosin. This figure represents the small mononuclear forms especially as greatly increased. Eosinophile cells and medullary cells, as well as nucleated red blood-corpuscles, are present in smaller number.

FIG. 6. **Acute Leukemia** in a child half a year old.—The number of white blood-corpuscles was only 48,000 to the cubic millimeter; nevertheless microscopic examination disclosed a leukemic state of the blood (nucleated red, slight increase in mononuclear and ~~eosinophile~~ cells). Clinically there existed marked enlargement of the spleen.

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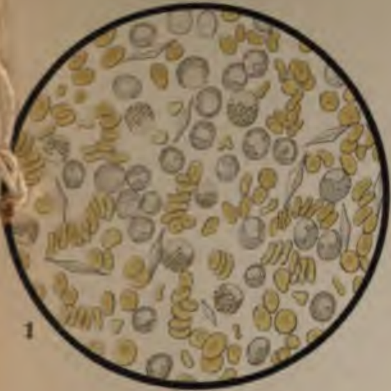

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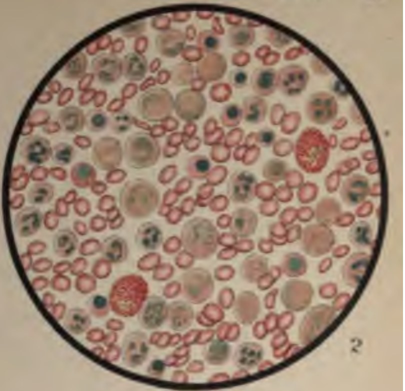

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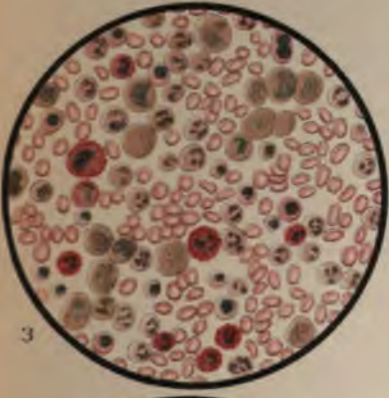

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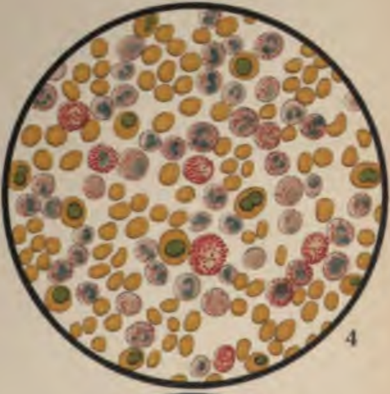

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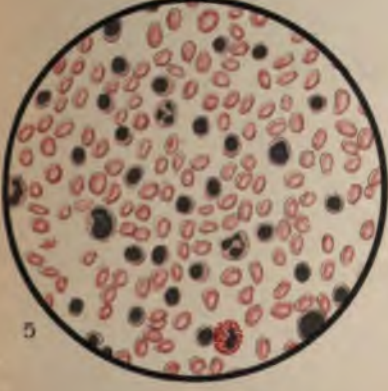

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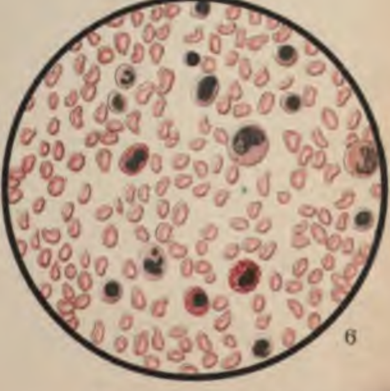

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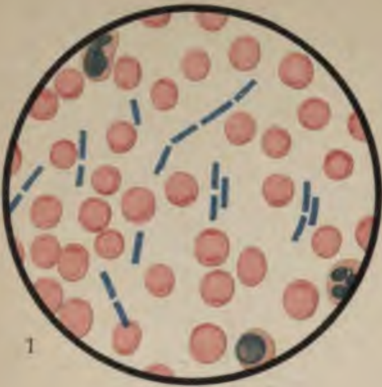

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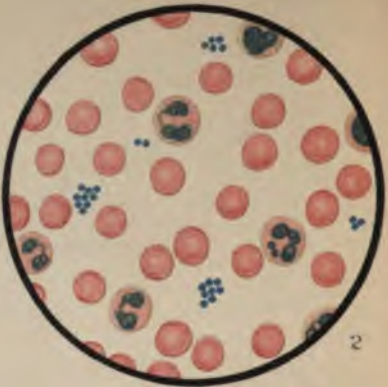

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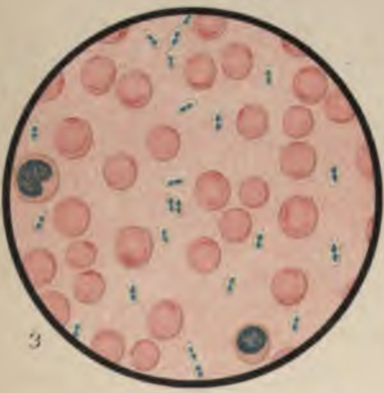

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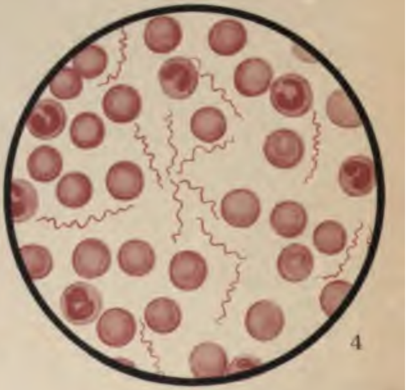

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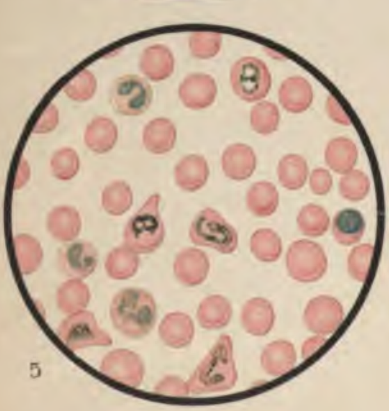

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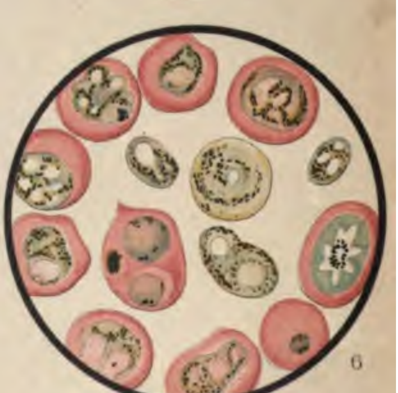

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PLATE 5.

PARASITES OF THE BLOOD.

FIG. 1. **Blood from a Subject dead of Anthrax.**—A full-grown man (brush-maker) died with symptoms of intestinal anthrax. The blood contained anthrax-bacilli in considerable numbers, singly and in chains.—Magnified 1000 times. Stained by Gram's method.

FIG. 2. **Blood from a Case of Pyemia.**—A child, five years old, died of multiple purulent osteomyelitis. During life yet the presence of staphylococcus pyogenes albus in the blood (removed with most scrupulous aseptic precautions) was demonstrated by culture and staining.

FIG. 3. **Pneumococci** in the blood of a mouse.—A mouse received subcutaneously half an exploratory syringeful of exudate from a case of fibrinous pleurisy (complicating pneumonia). Death resulted on the second day. In the blood were found large numbers of the diplococcus of pneumonia with distinct capsule. Stained by Gram's method.

FIG. 4. **Spirilla of Relapsing Fever.**—The blood of patients suffering from relapsing fever contains during the paroxysm numerous actively motile, delicate spirilla, stained here with magenta-red.

FIG. 5. **Blood from a Case of Malaria.**—Magnified 800 times. In a case of tertian intermittent fever malarial plasmodia were found included within red blood-corpuscles several hours before and also at the beginning of the paroxysm. The illustration shows five forms, which are not to be mistaken for leukocytes. The plasmodia lie within the bodies of the red cells, which they disintegrate. They contain yellowish and blackish granules of pigment (altered hemoglobin). Stained with methylene-blue and eosin.

FIG. 6. **Malarial Plasmodia.**—Stained with methylene-blue.—Magnified 1500 times. Various forms, some with spore-formation; also ectoglobular. (From the same case as Fig. 5.)

PLATE 6.

BLOOD-SPECTRA AND BLOOD-CRYSTALS.

FIG. 1, a. **Normal Solar Spectra,** with the various absorption-lines marked by letters (A, B, C, D, a, b, *a*). The blood changes the spectrum of the light passing through (marked dilution of the blood is necessary) in such a way that, in accordance with the behavior of the hemoglobin present, various portions of the colored spectrum are obliterated or absorbed. There thus appear at various places black bands of varying thickness.

b. Spectrum of blood rich in oxygen (oxyhemoglobin-spectrum) (two bands between D and E).

c. Spectrum of reduced hemoglobin.

d. Spectrum of methemoglobin (accompanying hemoglobinemia, destruction of the red blood-corpuscles through poisoning with potassic chlorate, pyrogallol, sulfonal, toadstools).

e. Spectrum of reduced CO-hemoglobin. The reduction accompanying carbon-monoxid poisoning is unattended with disappearance of the two bands between D and E; in contrast with reduced oxyhemoglobin (Fig. c).

f.–h. Spectra of hematin in acid and alkaline solutions and reduced (occurs in urine).

FIG. 2. **Hematoidin Crystals** (from old hemorrhagic focus).—Partly in rhombic plates, partly in granules.

FIG. 3. **Teichmann's Hemin-crystals.**—They serve for the demonstration of even slight traces of blood, old or recent. They are obtained by adding to the remnant of blood a crystal of sodic chlorid and a drop of glacial acetic acid, and effecting evaporation by gentle heat. Their recognition is of importance from a medico-legal point of view.

Tab. 6.

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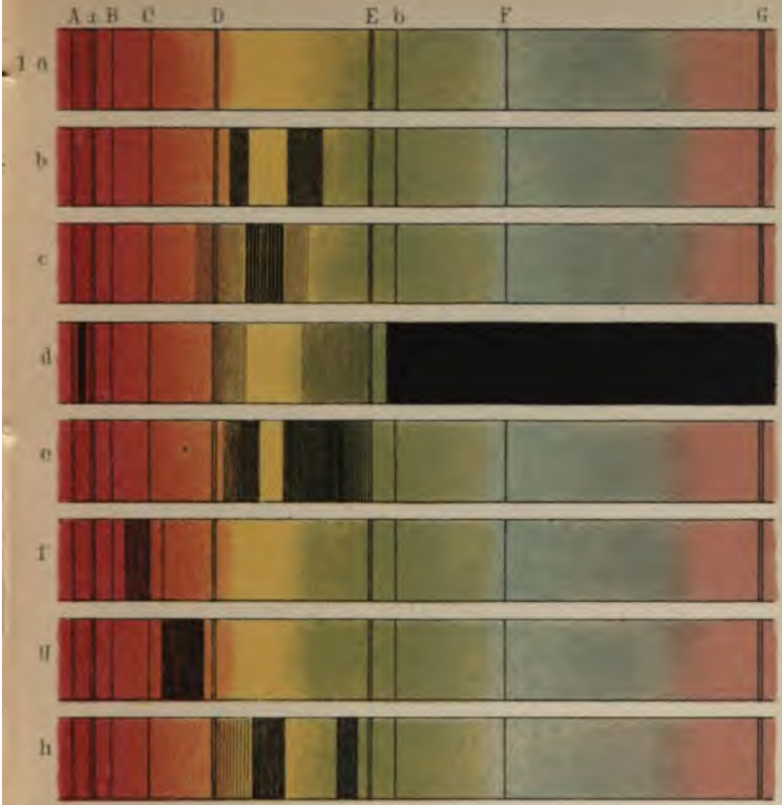

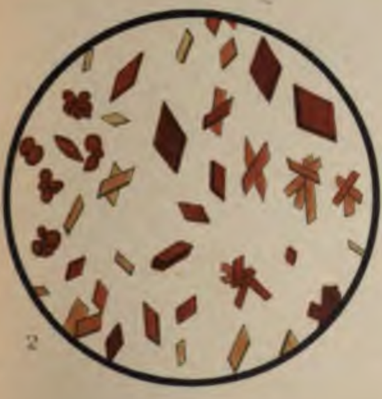

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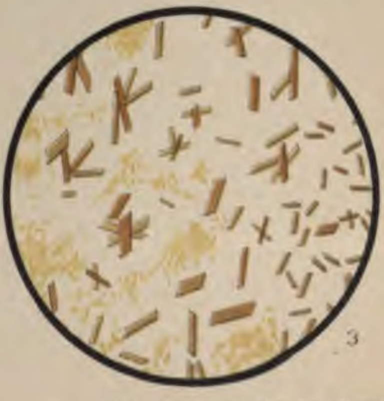

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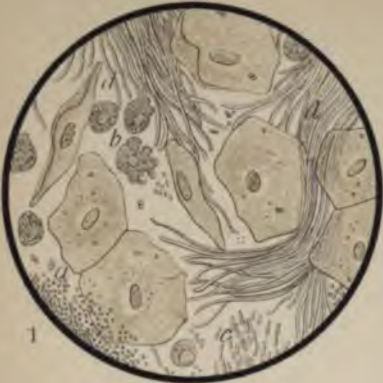

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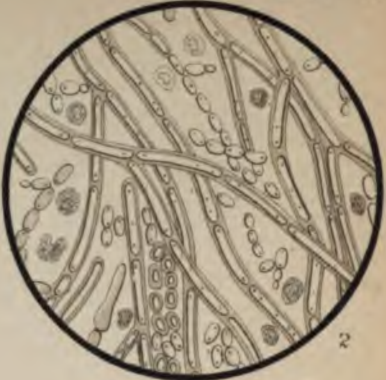

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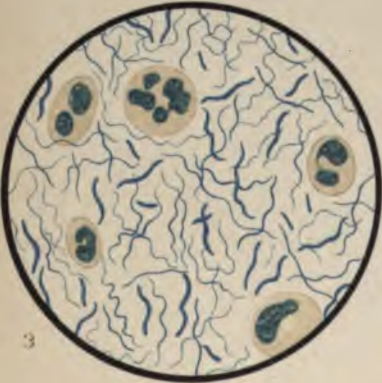

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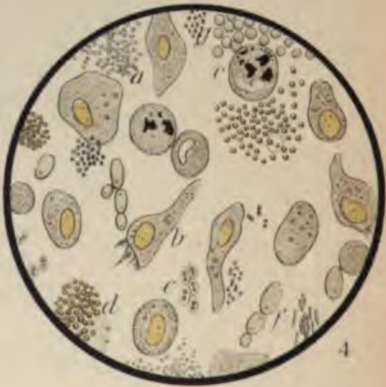

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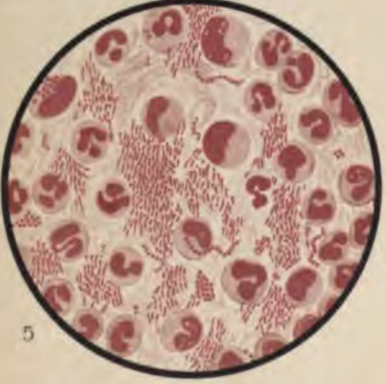

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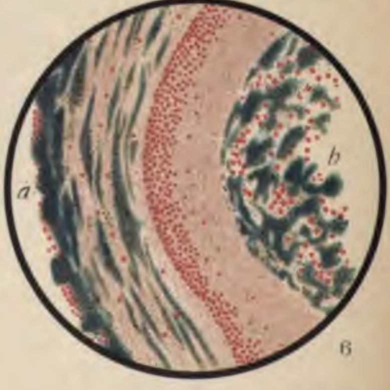

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PLATE 7.

MICROSCOPY OF THE MOUTH AND NASAL CAVITIES.

FIG. 1. **Gingival deposit** (unstained, with the addition of acetic acid).—There are present large squamous epithelial cells (*a*), partly covered by bacteria and granules of detritus; numerous white blood-corpuscles (*b*) in process of fatty degeneration: various bacteria (*c*): cocci, spirilla, bacilli, and especially large collections of a filamentous fungus: leptothrix bacillus (*d*), which bears an especial relation to decalcification of the teeth in connection with caries.—Magnified 350 times; as are also the following figures.

FIG. 2. **Thrush-fungus.**—This fungus (Oïdium albicans) is present especially, in nursing infants suffering from digestive disorders and in cachectic individuals. It forms a dense white deposit upon the mucous membrane of the mouth and pharynx. Microscopically it presents threads and spore-formation. It has been found also in abscesses.

FIG. 3. **Spirochetæ from a Case of Gangrenous Stomatitis.**—In a case of severe stomatitis actively motile spirilla (bacteria) were found in almost pure culture in two varieties (a coarse and a very delicate form) among the pus-corpuscles. Stained with gentian-violet.

FIG. 4. **Secretion from the Nasal Mucous Membrane.**—There are present large and small squamous epithelial cells (*a*), cylindric epithelium, with cilia (*b*), leukocytes filled partly with fat-drops, partly with carbon-particles (*c*), eosinophile cells (*d*), cocci, especially diplococci (*e*), budding fungi (*f*), spores of molds (*g*), such as aspergillus, mucor, etc., from the air.

FIG. 5. **Diphtheria-bacilli.** — (Streak-preparation, stained with magenta-red.) Derived from a diphtheric deposit upon the tonsils. Among the pus-corpuscles are nests of diphtheria-bacilli. These are slender rods, often somewhat thickened at the extremities (dumb-bell-shaped, club-shaped). The diagnosis must be rendered certain by culture. (See Epitome.) In addition to the specific excitants there are present, also, numerous other bacteria, streptococci (mixed infection), diplococci, and staphylococci.

FIG. 6. **Section through Diphtheria-membrane.** Stained with carmine and methylene-blue.—Magnified 50 times. At *a* is the surface of the false membrane. At *b* this is separated from the subjacent structures. The low power permits recognition of the masses of bacteria (blue) among the collections of cells and fibrin.

PLATE 8.

MICROSCOPY OF THE SPUTUM.

FIG. 1. **Normal Sputum** (unstained).—Magnified 350 times, as are the following figures. There are present squamous epithelium (*a*), cylindric epithelium (*b*), many leukocytes (*c*) imbedded in mucin threads, large cells filled with bright fat-granules (*d*), so-called alveolar epithelium (of doubtful origin, leukocytes ?), free myelin (*g*), isolated red blood-corpuscles (*e*), leukocytes filled with particles of carbon (soot-cells, *f*), free fat-drops (*h*), bacteria (*i*). ask.

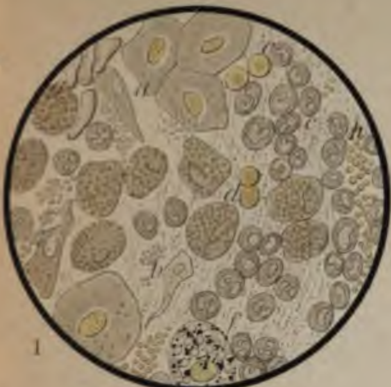
FIG. 2. **Normal Sputum.**—Stained with methylene-blue and eosin. The same picture as in Fig. 1. The nuclei of the cells, as well as the numerous bacteria of the sputum, appear more distinctly. An eosinophile cell is present. In several places is the so-called pulmonary sarcina, an innocuous coccus.

FIG. 3. **Pigment** (soot) **-cells** (on the left unstained, on the right stained with methylene-blue). The distended cells (leukocytes ?, alveolar epithelium ?) contain large drops of myelin, and among these black particles of carbon and dust.

FIG. 4. **Pigment** (cardiac lesion) **-cells** (unstained). In cases of pulmonary stasis (brown induration) resulting from cardiac lesions (mitral stenosis) the sputum contains characteristic large cells enclosing pigment-granules varying in color between yellow and dark brown (altered hemoglobin).

FIG. 5. **Sputum from an Attack of Asthma** (unstained).—In addition to the usual white blood-corpuscles there are present numerous leukocytes exhibiting bright, yellowish coarse granulations (*α*-granules); among these are numerous Charcot-Leyden asthma-crystals—colorless, pointed octahedra. In the middle of the field is a Curschmann spiral, a convoluted spiral formation made up of numerous individual filaments and originating in the smallest bronchioles (exudation ?). Disseminated throughout the field are numerous *α*-granules derived from ruptured eosinophile cells.

FIG. 6. **Sputum from a Case of Asthma.**—Stained with methylene-blue and eosin, and subsequently treated with potassic hydrate. The eosinophile cells appear distinctly, the disseminated (red) individual granules being readily distinguishable from the (blue) stained micrococci.



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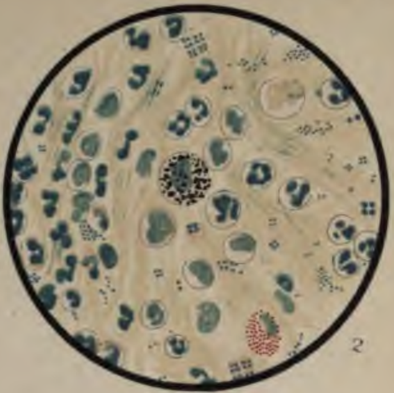

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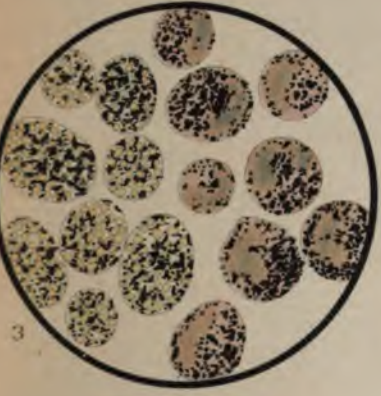

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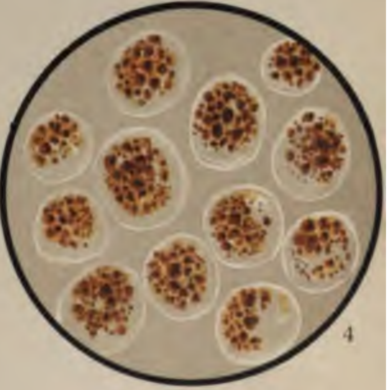

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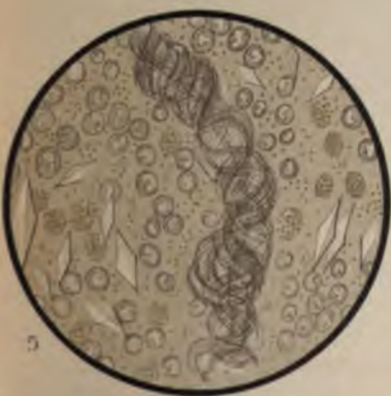

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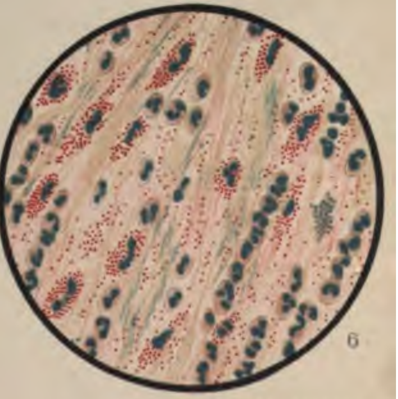

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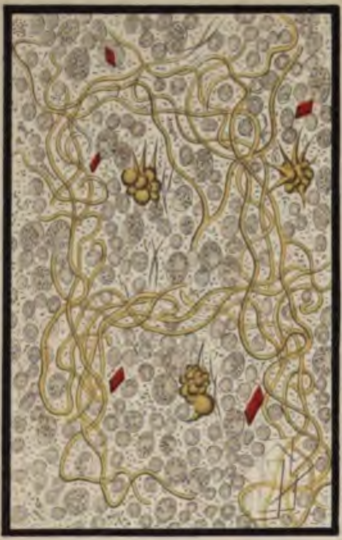

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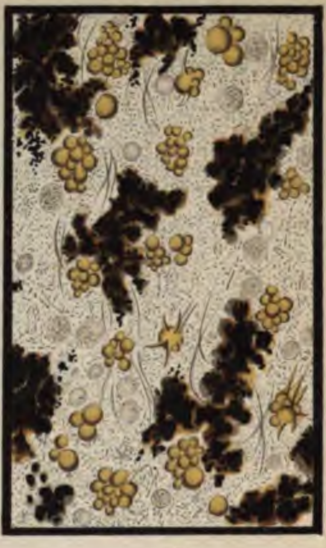

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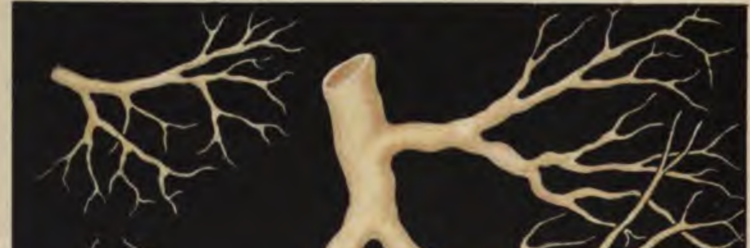

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PLATE 9.

Fig. 1. Sputum from an Abscess of the Lung.—The specimen was derived from a woman in whom a pulmonary abscess had developed in conjunction with a severe attack of pneumonia. After the rupture of the abscess there were discharged for a long time the following structures: elastic fibers (bright, yellowish filaments of double contour variously contorted), among which were large numbers of pus-corpuscles, for the most part in process of fatty degeneration; free fat-globules, large and small, containing fat-crystals (melting when heated), numerous fine fat-needles; bacteria; isolated hematoidin-crystals and cholesterin-crystals.

Fig. 2. Sputum from Gangrene of the Lung.—In the case of a man with chronic pulmonary tuberculosis there developed pulmonary gangrene (necrosis of a sequestrum of tissue). In the highly offensive, brownish, viscid sputum were found: a large amount of detritus (small granules and fragments), fat-drops, fat-crystals, much lung-pigment lying in large masses, pus-corpuscles in all stages of disintegration, and large numbers of bacteria.

Fig. 3. A large **Fibrinous Coagulum** (bronchial tree) from a case of diphtheria, coughed up through the cannula by a tracheotomized child. The preparation depicted in Plate 7, Fig. 6, was derived from this fibrinous tube, which represents a veritable cast of the bronchi. Above and to the left is shown a smaller coagulum, such as is sometimes expectorated in cases of pneumonia or fibrinous bronchitis. Smaller coagula are best observed by agitating the sputum in considerable water, the casts then unfolding.

PLATE 10.

THE PARASITES OF THE SPUTUM.

FIG. 1. **Pneumococci in Pneumonic Sputum.**—Among the blue-stained leukocytes and the red-stained red blood-corpuscles are numerous diplococci of Fraenkel exhibiting distinct capsule-formation, together with other cocci. The diplococci are found also in the expectoration of healthy individuals, although rarely in such large number.

FIG. 2. **Staphylococci from an Abscess of the Lung.**—In the diffluent pus discharged in large quantities in consequence of rupture of the abscess the staphylococcus pyogenes aureus (identity established by culture) was found in abundance. Stained with gentian-violet and eosin by Gram's method.

FIGS. 3 and 4. **Sputum from a Case of Tuberculosis.**—Treated with the specific stain (fuchsin and methylene-blue; see text), the sputum, spread in a thin layer and fixed on a cover-glass, shows on the left isolated, upon the right exceedingly numerous tubercle-bacilli. All other bacteria are stained blue. A reliable diagnostic conclusion can scarcely be reached from the number of bacilli, which are generally very abundant in the pus from cavities.

FIG. 5. **Sputum from a Case of Influenza-bronchitis.**—Stained with magenta-red. Among and within the pus-corpuscles are found the influenza-bacilli: delicate rods, mostly arranged in pairs and lying in groups of considerable size. The other bacteria are all plumper.

FIG. 6. **Sputum from a Case of Actinomycosis of the Lung.**—Stained with carmine and picric acid. Among the leukocytes can be seen granules that make up the microscopic yellow grains of the ray-fungus. These are found microscopically to consist of clubs (degenerated form) joined in the shape of a wreath or garland, and of threads or filaments (mycelium).

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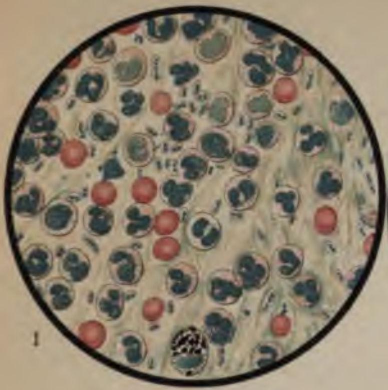
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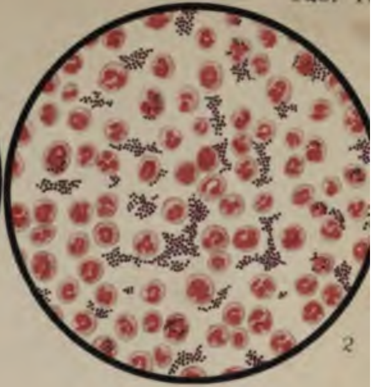
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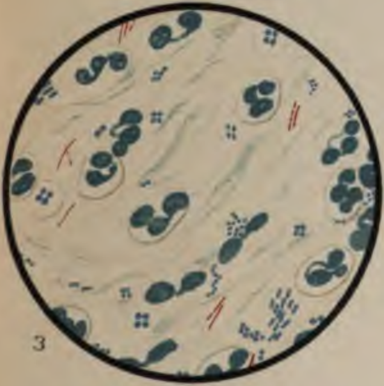
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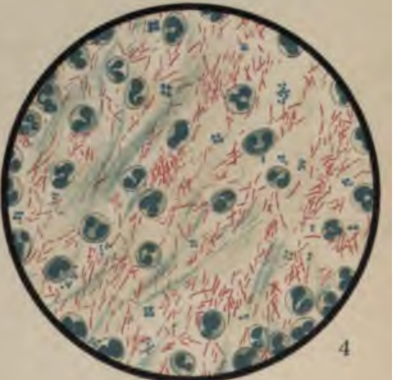
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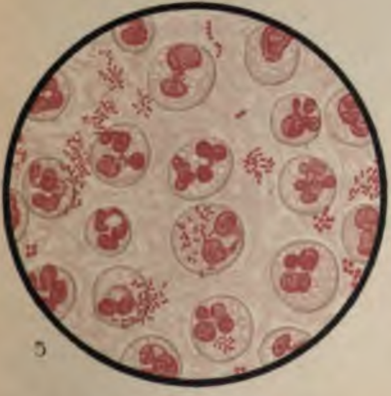
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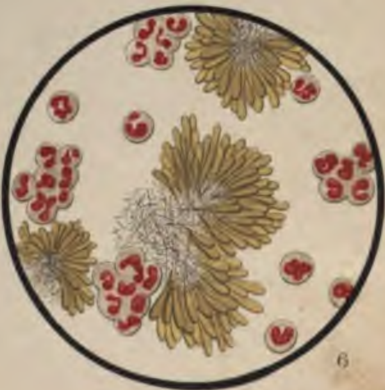
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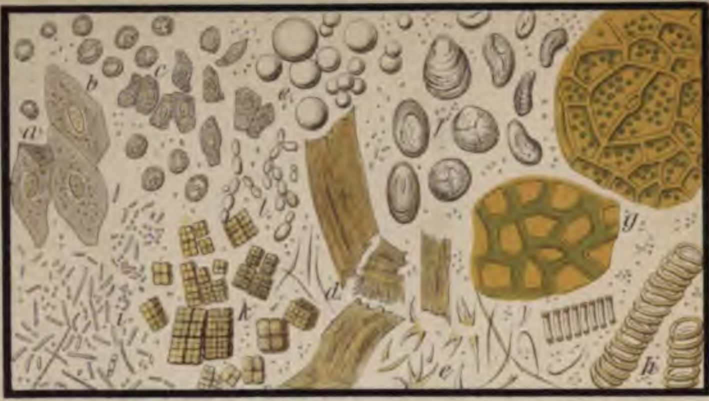

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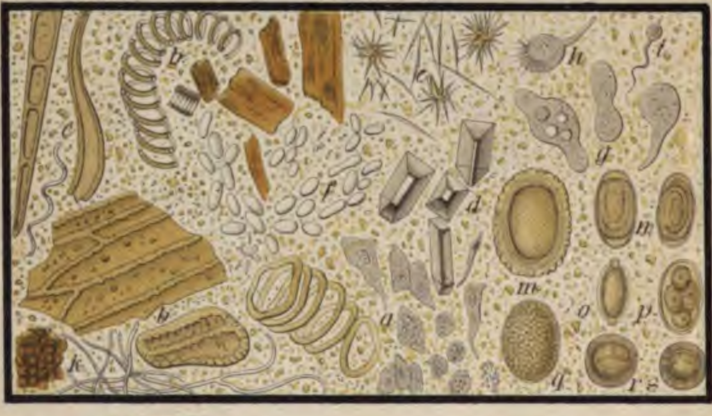

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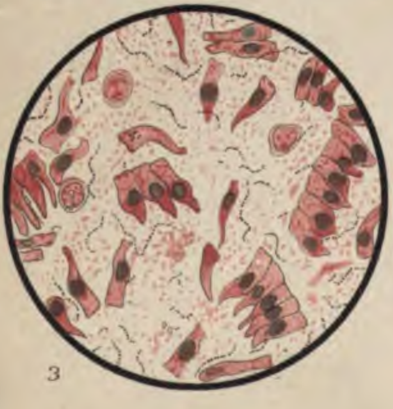

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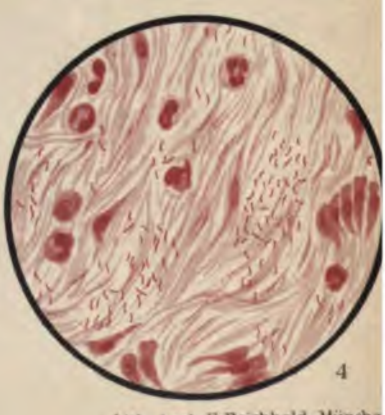

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PLATE 11.

MICROSCOPY OF CONTENTS OF STOMACH AND INTESTINES.

FIG. 1. **General View of Gastric Contents.**—In the vomited or siphoned matters are found: squamous epithelial cells (*a*) from the esophagus and the mouth; leukocytes (*b*); cylindric epithelial cells (*c*); particles of food, such as muscle-fibers (*d*), fat-drops and fat-crystals (*e*), starch-granules (*f*); chlorophyl-containing vegetable matters (*g*); spiral filaments (*h*); bacteria (*i*), especially gastric sarcina (*k*), cocci resembling bales of goods, which are sometimes present in abnormal numbers in conjunction with fermentative processes; budding fungi (*l*); at times also red blood-corpuscles, etc.

FIG. 2. **General View of the Feces.**—In the feces are found: desquamated epithelial cells, leukocytes (*a*); undigested vegetable matters, spiral filaments; stone-cells (*b*), cuticular formations (*c*); coffin-lid crystals (ammonio-magnesium phosphate, *d*); fat-crystals (*e*), especially numerous and forming distinct collections in acholic stools; yeast-fungi (*f*). At times are found, also, infusoria: ameba coli (*g*), which when present in increased numbers may induce disease of the bowel (amebic enteritis), trichomonas intestinalis (*h*); cercomonas intestinalis (*i*). Ova of the following worms are of rather common occurrence: ascaris (*m*), oxyuris (*n*), trichocephalus (*o*), ankylostomum (*p*), bothriocephalus (*q*), tænia saginata (*r*), tænia solium (*s*).

FIG. 3. **Intestinal Contents from a Case of Cholera Nostras.**—Stained by Gram's method. Derived from a man dead in eighteen hours from an attack of cholera morbus. In the preparation are seen immense numbers of desquamated intestinal cylindric epithelium, in part with distinct nuclei, and among them in pure culture streptococci pyogenes (etiologic relation?).

FIG. 4. **Intestinal Contents from a Case of Cholera Asiatica.**—Stained with magenta-red. Among the masses of epithelial cells there lie in nests imbedded in strands of mucus cholera-bacilli—slender rods more or less comma-shaped. (For cultural demonstration see Epitome, p. 27.)

PLATE 12.

THE MOST IMPORTANT COLOR-REACTIONS OF THE GASTRIC JUICE.

FIG. 1, *a* and *b*. When the gastric juice reddens blue *litmus-paper* (*b*)—that is, exhibits an acid reaction—it may contain: free hydrochloric acid, lactic acid, and other organic acids, acid salts.

FIG. 2, *a* and *b*. If red *Congo-paper* is stained bluish-black (*b*) by the gastric juice, only free hydrochloric acid or lactic acid is present.

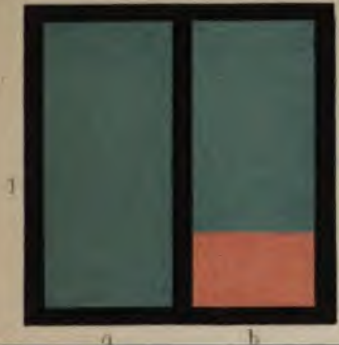
FIG. 3, *a* and *b*. If upon evaporation of the gastric juice in a porcelain dish, to which a few drops of *phloroglucin-vanillin solution* have been added, a distinct red ring appears, free hydrochloric acid is present; if the residue remains yellow, no free hydrochloric acid is present (anacidity).

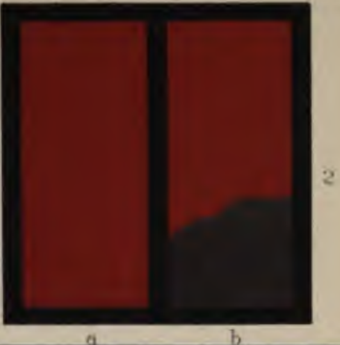
FIG. 4, *a* and *b*. If the gastric juice contains hydrochloric acid, the violet color of a dilute *methyl-violet solution* is converted into blue (*b*). (This test is not absolutely reliable.)

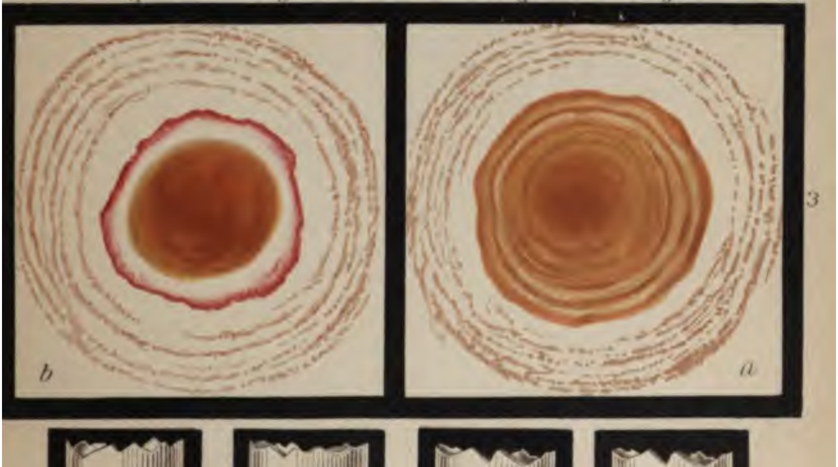
FIG. 5, *a* and *b*. If the gastric juice contains lactic acid, it will change the violet color of *Uffelmann's reagent* (1% solution of carbolic acid, with 2 drops of iron chlorid) into a distinct yellow (*b*). This test is more reliable if performed with an ethereal extract of the gastric juice (lactic acid being soluble in ether).

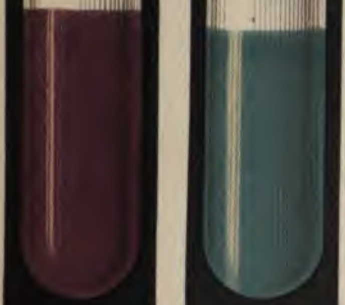
(For details see Epitome, p. 82, et seq.)

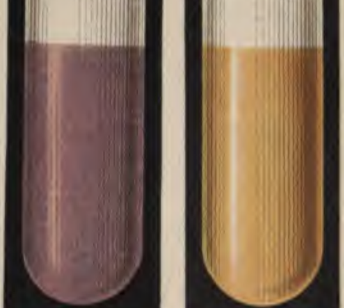
Tab. 12.











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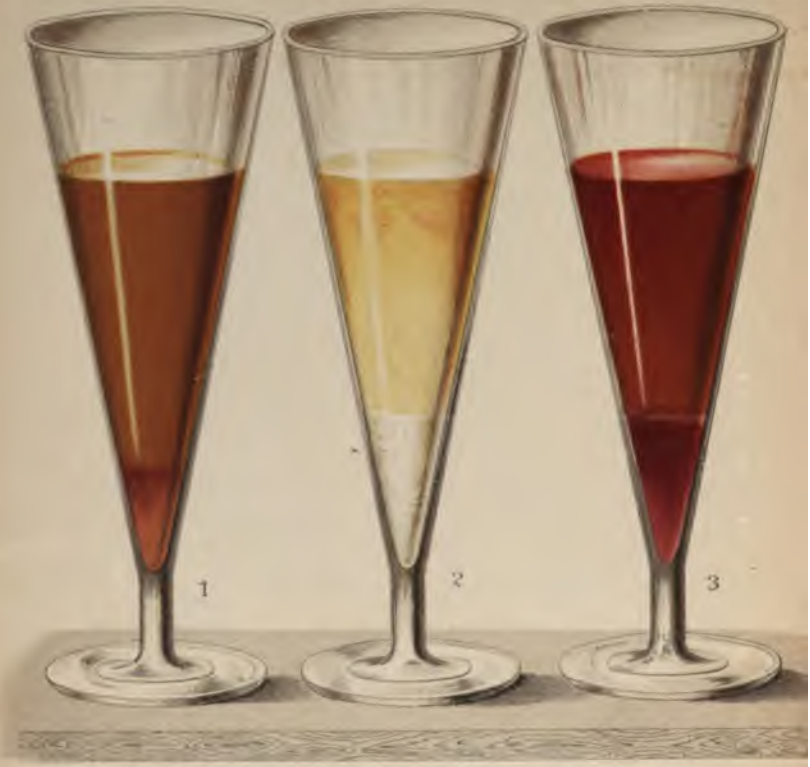

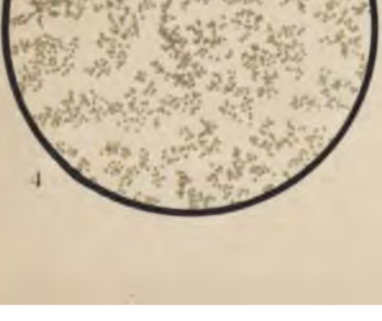
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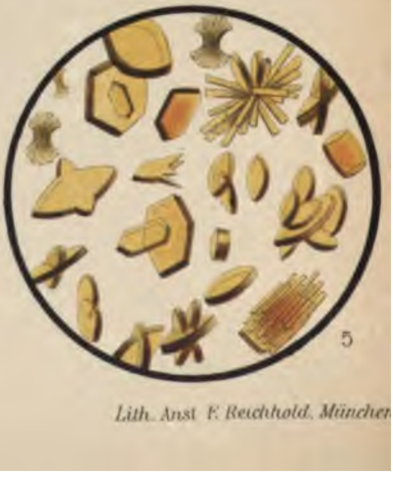
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PLATE 13.

URINARY SEDIMENTS.

The most important macroscopic varieties of urinary sediment are represented in the three conical glasses.

FIG. 1. **Brick-dust Sediment.**—This is formed only of uric acid, and is found in abundance in febrile states, after active bodily exertion, etc. It dissolves on heating.

FIG. 2. **Yellowish Friable Sediment.**—This may consist of phosphates (soluble on addition of acid), of pus-corpuscles, of renal elements, of bacteria (demonstrable microscopically).

FIG. 3. **Bloody Sediment.**—Demonstrable by Heller's blood-test (see Plate 20, Fig. 10), as well as by microscopic examination (renal or vesical hemorrhage, hemoglobinuria).

Uric-acid Sediment.

FIG. 4. **Sodium Urate**, in small yellowish granules, frequently adherent to other elements, especially casts, etc.

FIG. 5. **Uric-acid Crystals**, varying in color from yellow to yellowish-brown, in large, not entirely regular plates, in whetstone, dumb-bell, and comb-like form and arrangement.

Both sediments occur only in urine of acid reaction, and are precipitated by addition of acid. They dissolve upon addition of potassium hydroxid.

PLATE 14.

CRYSTALLINE URINARY SEDIMENTS.

FIG. 1. **Calcium Oxalate.**—This is found in acid urine, especially in the characteristic form of an envelop, as large, but more frequently as quite small, glittering crystals. It is readily soluble in hydrochloric acid, insoluble in acetic acid. It is especially abundant in cases of oxaluria (but quantitative determination is necessary to verify the diagnosis).

FIG. 2. **Hippuric Acid.**—This occurs but seldom in the urine (after ingestion of benzoic acid or after eating bilberries, etc.) and is without significance.

FIG. 3. **Ammonio-magnesium Phosphate, Triple Phosphate.**—This appears as large rhombic columns (coffin-lid) and constitutes a common sediment in feebly acid or alkaline urine.

FIG. 4. **Ammonium Urate.**—This consists in the recent state of tufts or sheaves of fine, densely compressed needles. The needles are gradually so transformed as to appear as plump spheres with isolated needles projecting from the surface (thorn-apple form). Ammonium urate is common in alkaline urine (ammoniacal fermentation).

v FIG. 5. **Calcium Carbonate.**—This occurs in large spheres, often dumb-bell-shaped, or in small granules, which dissolve upon the addition of acetic acid, with effervescence (free CO_2).

FIG. 6. Upper portion: **Calcium Sulphate**; lower portion: **Calcium Phosphate** (without significance).

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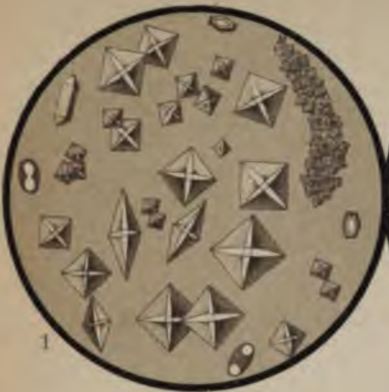
Tab. 14.



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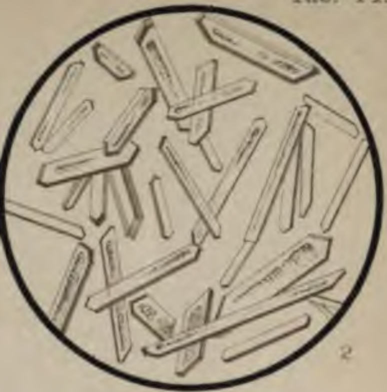

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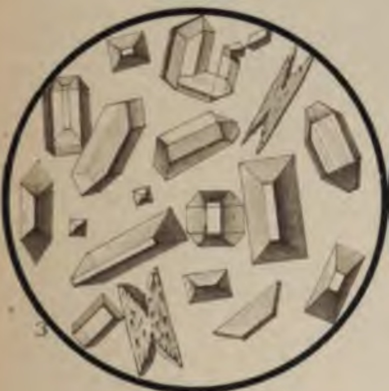

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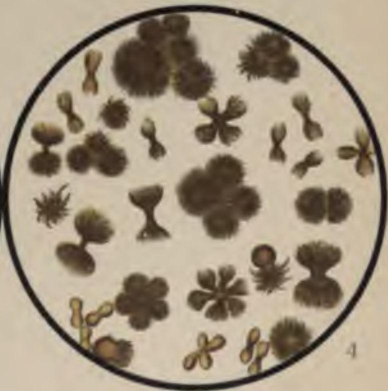

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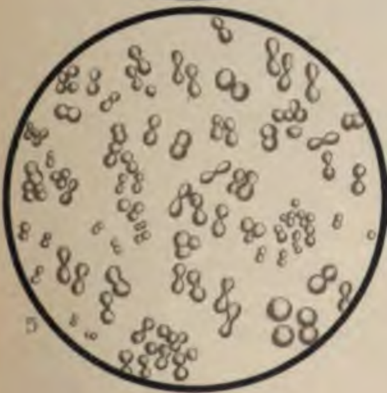

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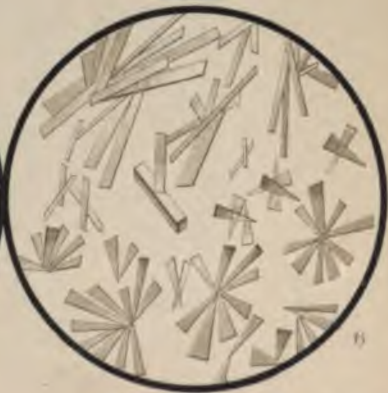

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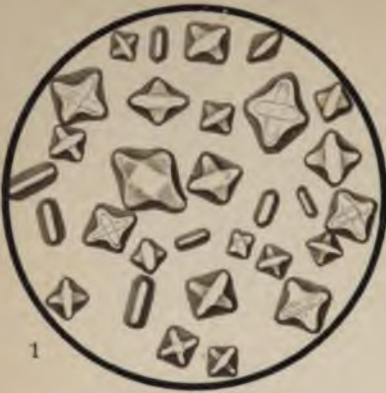

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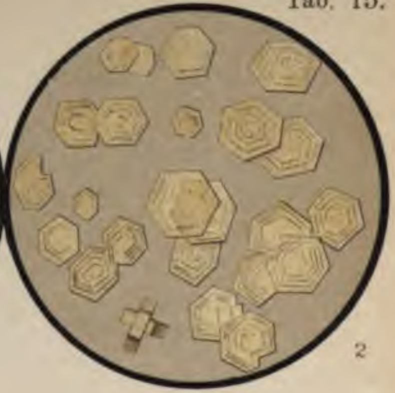

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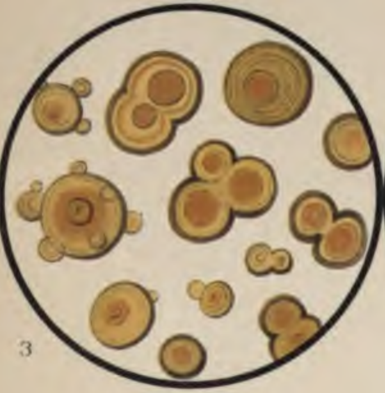

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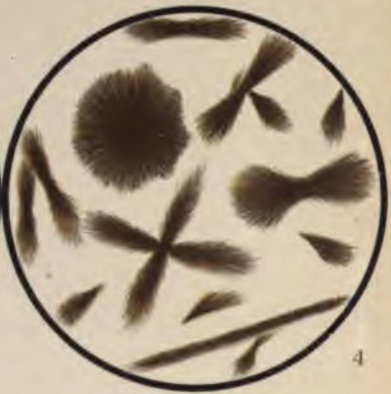

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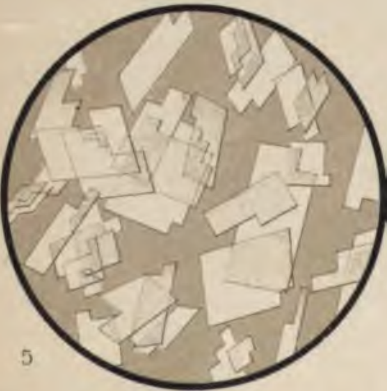

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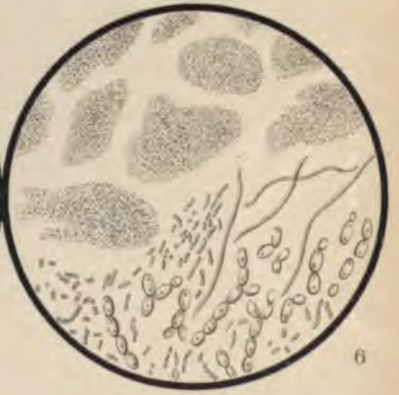

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PLATE 15.

CRYSTALLINE URINARY SEDIMENTS.

FIG. 1. **Basic Magnesium Phosphate.**—The rhombic plates are soluble in acetic acid; the crystals are not common. v. Ca)

FIG. 2. **Cystin.**—The regular six-sided plates are insoluble in acetic acid, but soluble in ammonia. Cystin burns with a bluish-green flame. It occurs in cases of periodic cystinuria and of articular rheumatism (without significance).

FIG. 3. **Leucin.**—The large or small yellow spheres form upon evaporation of the urinary sediment. Leucin occurs always in association with tyrosin. Both are terminal products of albuminous metabolism (amido-acids).

FIG. 4. **Tyrosin.**—This crystallizes in tufts or sheaves of fine or coarse needles and is soluble in ammonia.

Leucin and tyrosin are found in the urine in cases of acute yellow atrophy of the liver, of phosphorus-poisoning, and of some infectious diseases, etc.

FIG. 5. **Cholesterin.**—The fine plates occur but seldom; among other conditions in association with echinococcus of the kidney, with filaria, with cystitis.

FIG. 6. In the upper half of the field is shown an amorphous, finely granular sediment of **Earthy Phosphates** (calcium and magnesium). This is soluble in acetic acid, without effervescence (as contrasted with calcium carbonate). In the lower half are found the various bacteria of urinary fermentation: budding fungi, bacilli, collections of micrococci.

PLATE 16.

ORGANIZED URINARY SEDIMENTS.

FIG. 1. **Squamous Epithelium from Urethra and Bladder.**—The superficial layers of the bladder contain large squamous epithelial cells (*a*), the deeper layers club-shaped cells with tenuous extremities (*b*). Frequently the cells are covered with bacteria.

FIG. 2. **Epithelial Cells from the Pelvis of the Kidney.**—Epithelial cells with tenuous extremities and large nuclei. They are not to be differentiated with certainty from similar cells derived from the bladder.

FIG. 3. **Renal Epithelium.**—Characteristic cubic cells with large distinct nuclei, often involved in all stages of fatty degeneration. They frequently form deposits upon renal tube-casts. Their presence in large numbers is significant of profound disease of the kidney.

FIG. 4. **Pus-cells.**—Small, round cells, with indistinct nuclei, often collected in groups, often filled with fat-granules, often seated upon tube-casts.

FIG. 5. **Red Blood-corpuscles.**—In all stages of contraction or distention if not recent. By dissolution of the hemoglobin there result the so-called shadows with double contour. The red cells often assume a thorn-apple form.

FIG. 6. **Spermatozoa.**—Found not rarely in urinary sediment (with especial frequency in cases of spermatorrhea). At *a* are seen so-called Böttcher crystals; at *b* prostate granules; at *c* a testicular cast (from the prostate).

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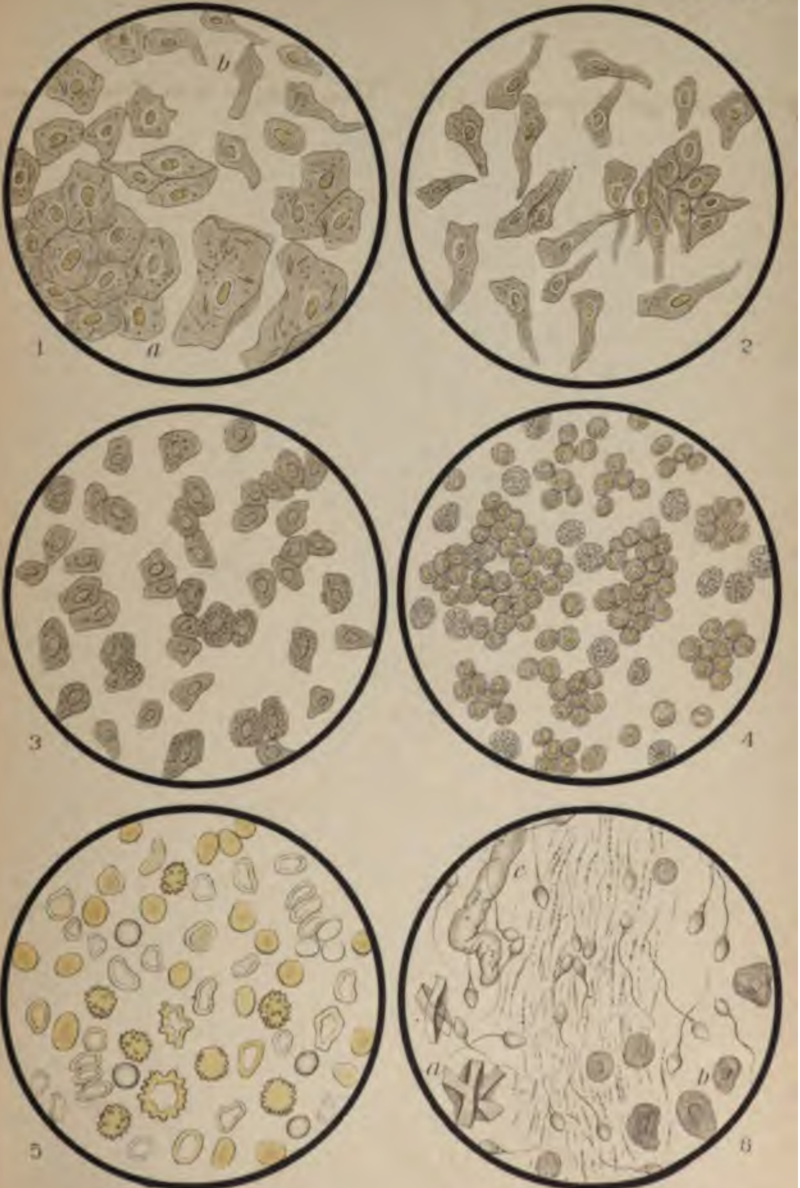

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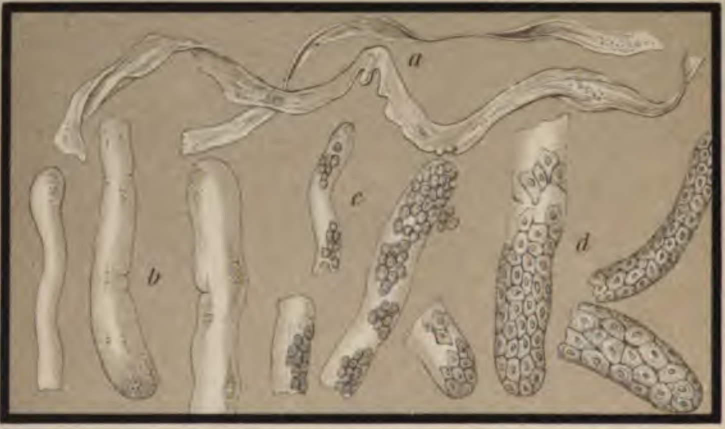

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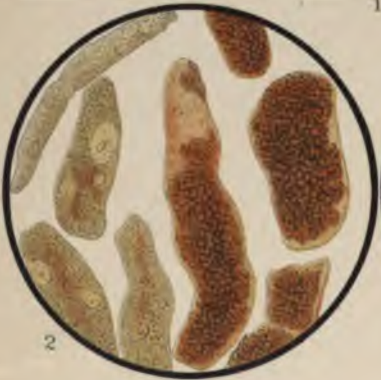

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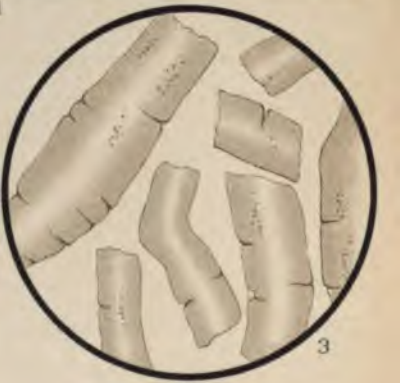

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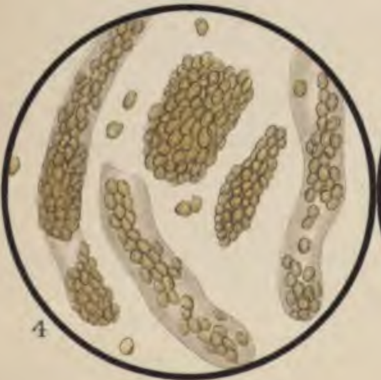

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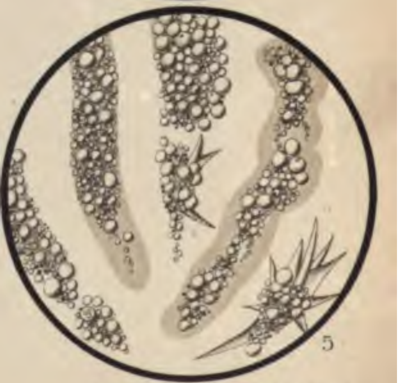

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PLATE 17.

URINARY TUBE-CASTS.

FIG. 1. In the upper portion of the figure are shown **Cylindroids** (*a*), which are without significance. Below are **Hyaline Tube-casts** (*b*), which occur in conjunction with all diseases of the kidney (inflammation, stasis, irritation by toxins), in the form of narrow or broad cylinders. They occur as the result of a form of exudation into the uriniferous tubules. They are frequently the seat of white blood-corpuscles (*c*), or of renal epithelium (*d*). The latter relation is significant of profound disturbance.

FIG. 2. **Coarsely and Finely Granular Tube-casts.**—These result mostly from the transformation of superposed red and white blood-crystals (degeneration). They occur frequently in the course of acute inflammations of the kidney.

FIG. 3. **Waxy Tube-casts.**—These have a sharper contour and a more transparent appearance than hyaline tube-casts. They are found more commonly in cases of chronic nephritis.

FIG. 4. **Blood-casts.**—Narrow or broad tube-casts densely covered with red blood-corpuscles. They are found in conjunction with hemorrhagic inflammations of the kidney.

FIG. 5. **Fatty Casts.**—Tube-casts densely covered with large and small fat-granules and also fat-crystals occur, among other conditions, in especial abundance in association with a certain form of chronic nephritis (large white fatty kidney).

PLATE 18.

URINARY SEDIMENTS ATTENDING DISEASES OF THE BLADDER AND THE KIDNEYS.

FIG. 1. **Sediment from Cystitis.**—The very abundant sediment contains: numerous epithelial cells from all layers of the bladder (in accordance with the severity of the affection); numerous pus-corpuscles, frequently collected in masses; isolated red blood-corpuscles; many bacteria; coffin-lid and ammonium-urate crystals.

FIG. 2. **Sediment from Calculous Pyelitis.**—There are present numerous epithelial cells, especially also cells with tenuous extremities, numerous pus-corpuscles (pus-casts), a considerable number of red blood-corpuscles, uric-acid crystals.

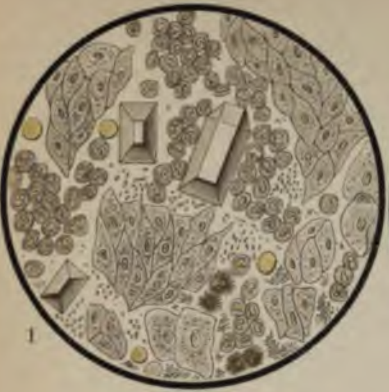
FIG. 3. **Sediment from Acute Hemorrhagic Nephritis.**—Abundant sediment of hyaline casts and epithelial tube-casts, white and red blood-corpuscles free and seated upon casts. No fattily degenerated cells.

FIG. 4. From the same disease after the lapse of two weeks. The hemorrhagic character has somewhat receded. There are present also granular and waxy casts and fattily degenerated epithelial cells.

FIG. 5. **Chronic Nephritis.**—Large, red, variegated kidney. Numerous tube-casts, with leukocytes and renal epithelial cells attached, blood-casts, renal epithelial cells (not fattily degenerated), red and white blood-corpuscles.

FIG. 6. **Chronic Nephritis.**—Large, white kidney. Numerous fatty granular casts, fattily degenerated epithelial cells (granule-cells), white blood-corpuscles, free fat-drops.

Tab. 18.



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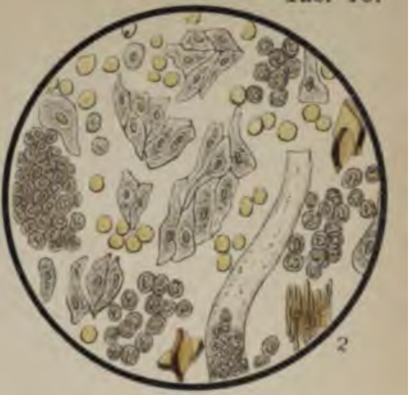

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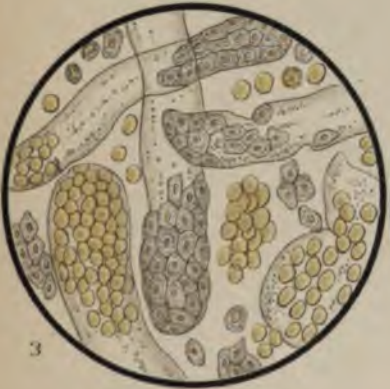

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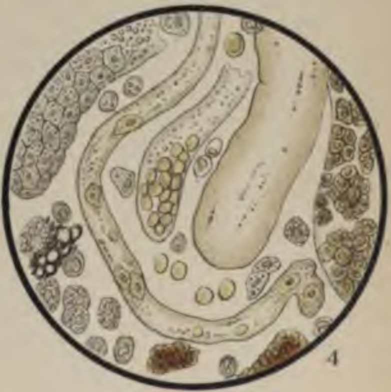

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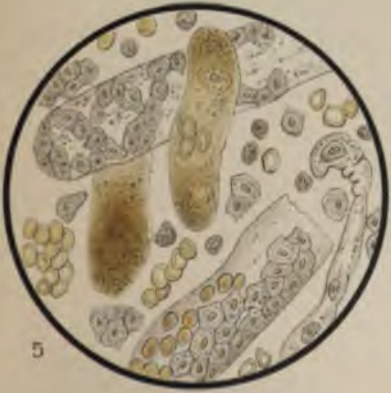

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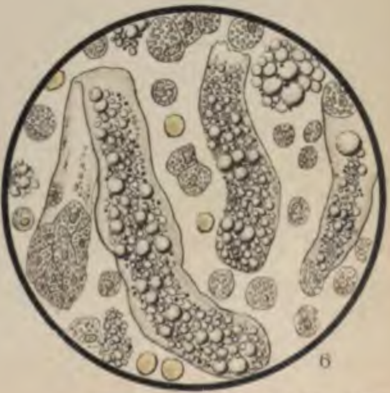

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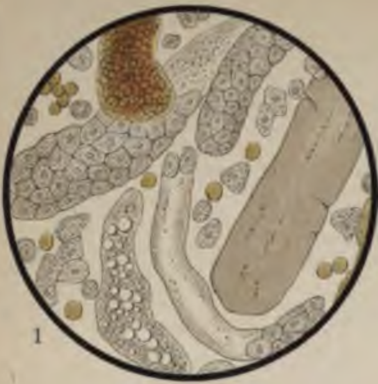
Tab. 19.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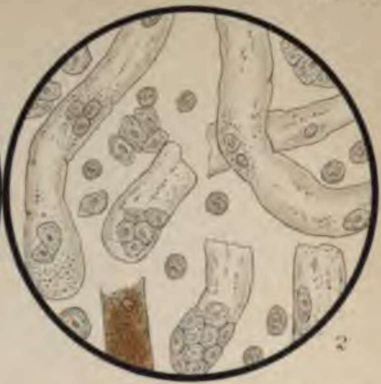
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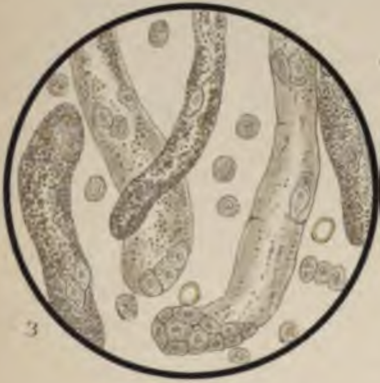
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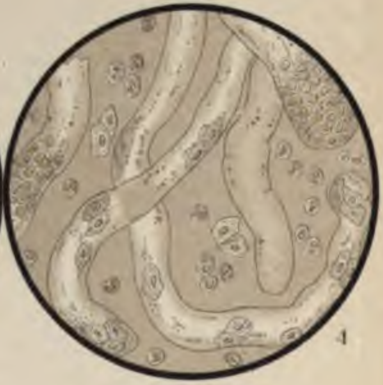
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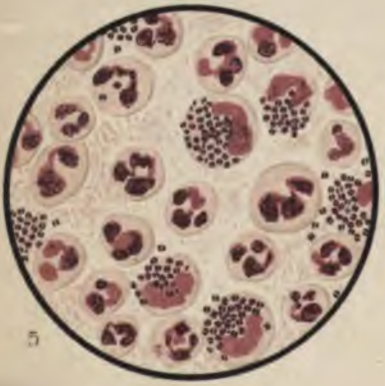
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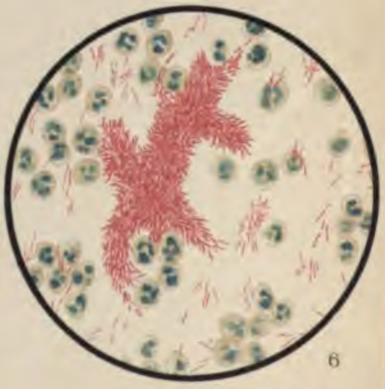
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PLATE 19.

FIG. 1. **Sediment from Chronic Parenchymatous Nephritis.**—This consists of casts, some hyaline, some with cells attached (renal epithelium), some granular, some waxy; renal epithelial cells (also fattily degenerated); white blood-corpuscles.

FIG. 2. **Sediment from True Contracted Kidney.**—The amount is scanty (centrifuge) and the deposit contains some hyaline casts, some epithelial casts, some renal epithelial cells, and white blood-corpuscles.

FIG. 3. **Sediment from Febrile Albuminuria.**—There are present in considerable number hyaline tube-casts, some with sodium urate attached, and also white blood-corpuscles.

FIG. 4. **Sediment from Amyloid Kidney.**—This contains (in small amount) hyaline tube-casts, some with leukocytes attached, some renal epithelial cells, and white blood-corpuscles.

FIG. 5. **Pus from Gonorrhea.**—Stained with gentian-violet. Partly enclosed in individual leukocytes, partly free among them, are found the characteristic collections of gonococci (biscuit-form).

FIG. 6. **Pus from Tuberculous Cystitis.**—In addition to individual tubercle-bacilli there is present a dense collection of bacilli. When the bacilli are not numerous the Dahmen procedure is of advantage. (See Epitome, Section II., p. 26.)

PLATE 20.

THE MOST IMPORTANT COLOR-REACTIONS OF THE URINE.

FIGS. 1 to 3. **Trommer's Test for Sugar.**—Potassium hydroxid and copper sulphate.

FIG. 1. Urine free from sugar does not dissolve copper sulphate and assumes a greenish-yellow color on boiling.

FIG. 2. Urine containing sugar dissolves the hydrated cupric oxid formed, with the development of a blue color, and precipitates on heating hydrated cuprous oxid in yellowish-red clouds (Fig. 3)—reduction-process.

FIG. 4. **Bismuth-test.**—Addition of Nylander's solution (see Epitome, Section III, p. 108). On heating, metallic bismuth is precipitated in black clouds if sugar be present.

FIG. 5. **Moore's (Caramel-) Test.**—If to urine containing sugar is added one-third the quantity of potassium hydroxid and heat applied (for three minutes), a chestnut-brown color results.

FIG. 6. **Ferric-chlorid Reaction in Diabetes.**—This consists in the development of a Bordeaux-red color when diacetic acid is present in the urine, and is thought to indicate threatening diabetic coma [?].

FIG. 7. **Peptone-test.**—When albumoses, etc., are present in the urine the addition of potassium hydroxid and solution of copper sulphate in the cold is followed by the development of a violet color.

FIG. 8. **Indican-test.**—If urine and pure hydrochloric acid be mixed in equal parts, and calcium hypochlorit in solution be added drop by drop, any indoxyl present will be oxidized into blue indigo (various intestinal disorders, fermentative processes). The mixture may be further shaken with chloroform or ether.

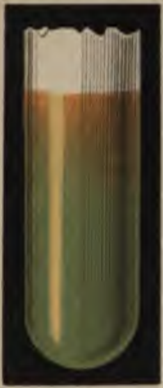
FIG. 9. **Test for Biliary Coloring-matter.**—On shaking with chloroform the urine from a case of jaundice the fluid assumes a yellow color (bilirubin).

FIG. 10. **Heller's Blood-test.**—On the addition of one-third potassium hydroxid and boiling, the precipitated phosphates carry the blood coloring-matter with them to the bottom in the form of red clouds.

FIG. 11. **Test for Melanin.**—In cases of melanotic sarcoma the urine treated with iron chlorid assumes a deep-black color.

FIG. 12. **Diazo-reaction.**—In cases of typhoid fever, tuberculosis, etc., the addition of a mixture of sulphanilic acid and sodium nitrite (see Epitome, p. 113) gives rise to the development of a bright-red color, apparent also in the froth on shaking.

Tab. 20.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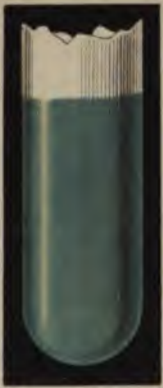
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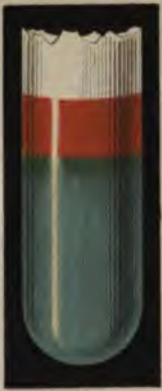
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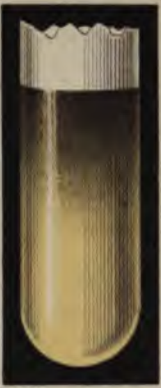
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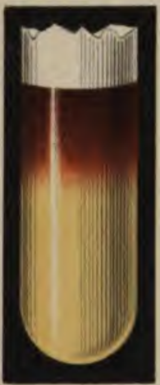
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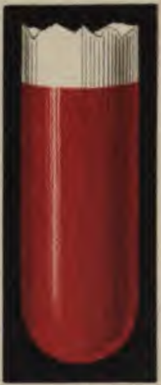
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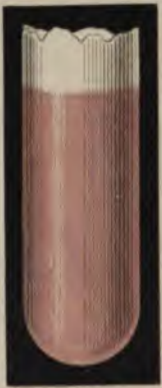
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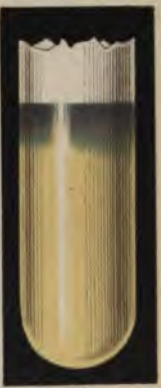
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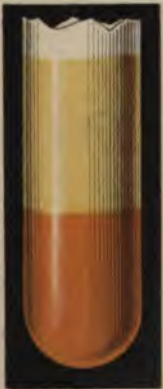
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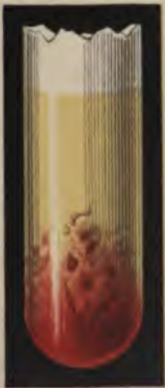
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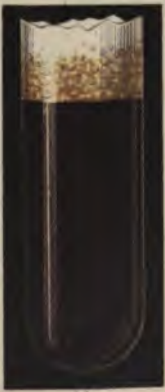
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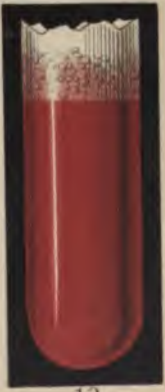
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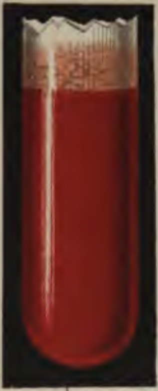
Tab. 21.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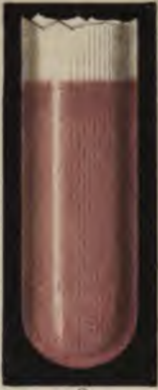
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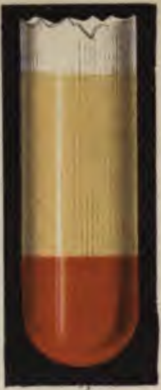
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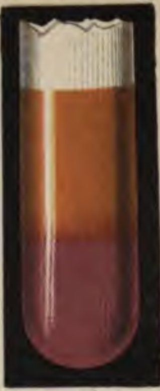
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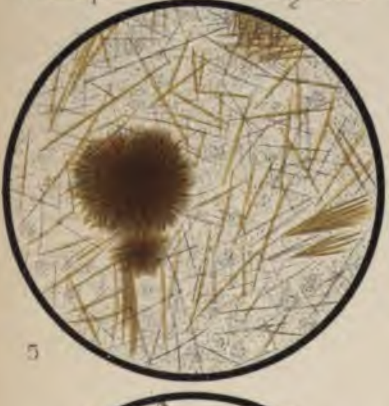
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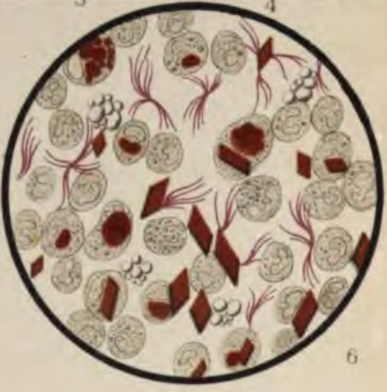
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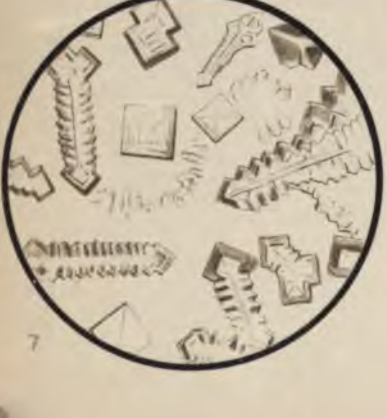
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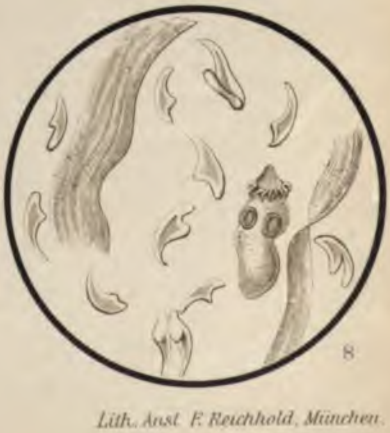
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PLATE 21.

DEMONSTRATION OF SOME MEDICAMENTS IN THE URINE.

FIG. 1. Demonstration of **Antipyrin in the Urine.**—On addition of iron chlorid a brownish-red color results.

FIG. 2. Demonstration of **Salicylic Acid.**—Iron chlorid induces an intense violet color.

FIG. 3. Demonstration of **Bromin** (potassium bromid).—Addition of fresh chlorin-water and agitation with chloroform yield a yellow color.

FIG. 4. Demonstration of **Iodin** (potassium iodid).—Addition of several drops of fuming nitric acid and agitation with chloroform yield a reddish-violet color.

Contents of Cysts, Abscesses, etc.

FIG. 5. **Crystals of Uric Acid from a Gouty Nodule.**—In a ruptured gouty nodule there were found in large numbers the crystals of uric acid depicted, suggestive of fat-needles, from which they differed, however, in their reactions.

FIG. 6. **Hematoidin-crystals from an Old Abscess.**—Among the profoundly fattily degenerated pus-corpuscles and fat-drops there were found large numbers of hematoidin-crystals, partly free, partly included in cells, and partly in the form of red needles.

FIG. 7. **Echinococcus-fluid** (evaporated).—There remain numerous crystals of sodium chlorid.

FIG. 8. **Echinococcus-fluid** (sediment).—There are present numerous echinococcus-hooklets, portions of membrane (exhibiting characteristic lamination), and a calcified scolex.

PLATE 22.

THE MOST IMPORTANT PYOGENIC MICROORGANISMS.

FIG. 1. **Staphylococcus Pyogenes Albus** from an abscess of the parotid gland, complicating a case of typhoid fever (secondary infection).

FIG. 2. **Streptococcus Pyogenes** in the pus from a case of empyema secondary to pneumonia.

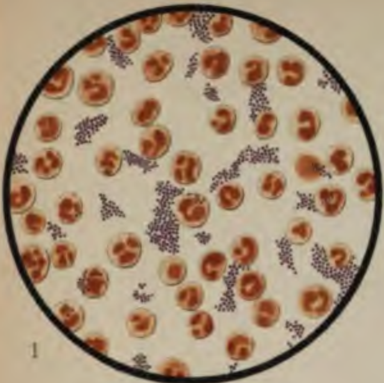
FIG. 3. **Bacterium Coli Commune** in pus from a subphrenic abscess.

FIG. 4. **Proteus Vulgaris**, together with other bacteria, from putrid perforation-peritonitis.

FIG. 5. **Bacillus of Glanders** from pus evacuated from a cervical abscess.

FIG. 6. **Typhoid Bacilli.**—Streak-preparation from the spleen of a subject dead of typhoid fever.

Tab. 22.



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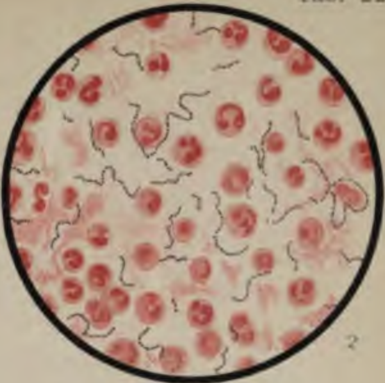

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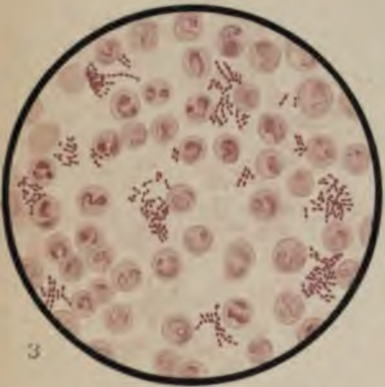

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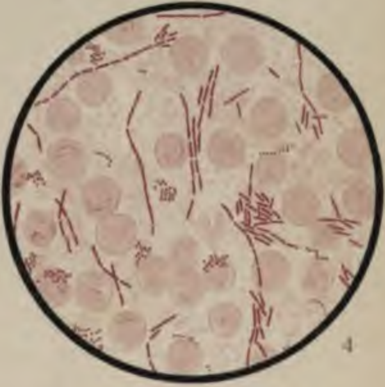

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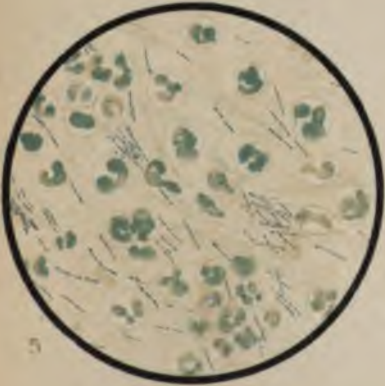

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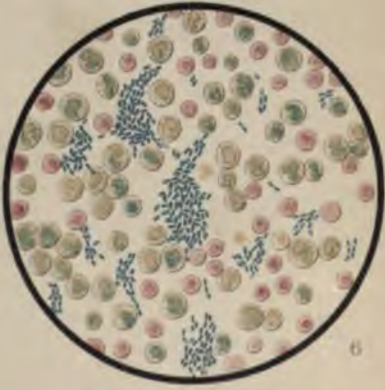

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NORMAL PROJECTION OF THE VISCERA

AND

PERCUTORY TOPOGRAPHY.

(PLATES 23-28.)

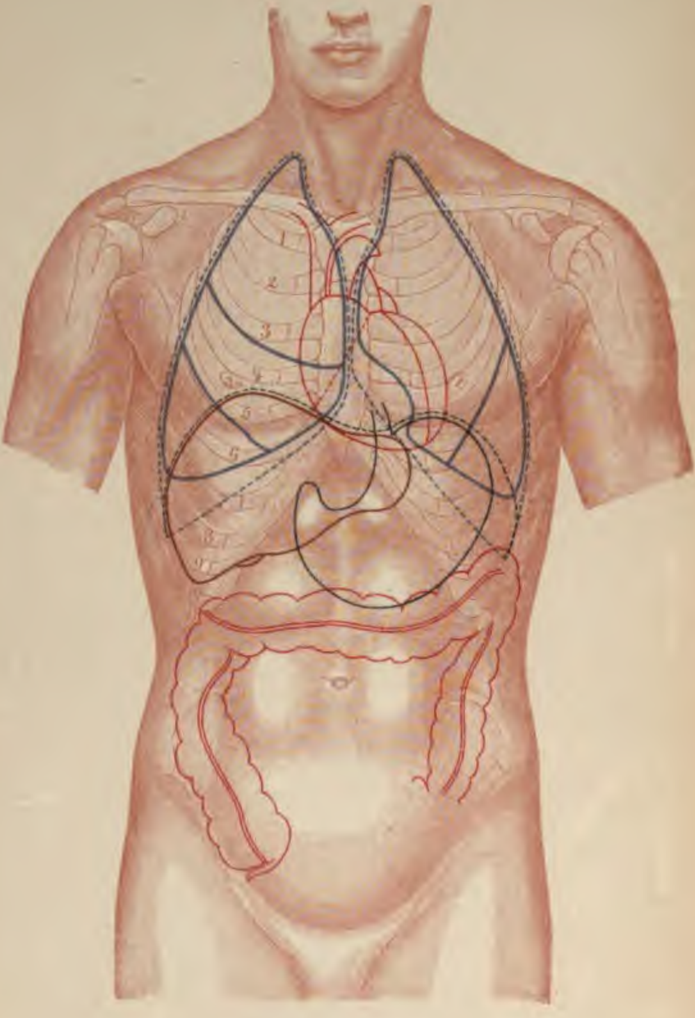

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PLATE 23.

PROJECTION OF THE INTERNAL ORGANS UPON THE ANTERIOR ASPECT OF THE BODY.

1. The **Lungs** (blue outline).—The course of the pleuræ (interrupted blue line) is readily visible. The pleuræ project beyond the lower margins of the lungs on both sides (greatest separation in the axillary line 10 cm.), on the left also in the region of the heart, thus forming the complementary spaces.

The **Right Lung** has three lobes: the upper extends to the upper border of the fourth rib, the middle from this point to the upper border of the sixth rib, with the lower lobe to the outside.

The **Left Lung** has two lobes: the upper extends to the upper border of the sixth rib, with the lower at its outer side.

2. The **Heart** (red outline).—The base of the heart (above) corresponds with the second intercostal space. The right border (covered by lung) projects beyond the right sternal margin; the left border lies slightly within the mammillary line. The apex-beat of the heart is situated in the fifth intercostal space on the left. Only a portion of the right ventricle (incisura cardiaca) is in contact with the chest-wall, the remainder being covered by lung. The origin of the aorta is at the base beneath the upper portion of the sternum. To its right is the superior vena cava, to its left the pulmonary artery. Only the margin of the left ventricle and auricle is to be seen from in front; the remainder faces backward.

3. The **Diaphragm** (interrupted black line).—It stands at a slightly higher level on the right than on the left, and at a varying level in accordance with respiration. Its highest point in the cadaver is at the level of the attachment of the fourth rib.

4. The **Liver** (brown outline).—The position of both lobes is self-evident. The gall-bladder is situated at a point where the lower margin of the liver intersects the costal arch (somewhat within the right mammillary line).

5. The **Stomach** (black outline).—The cardia lies behind the attachment of the left seventh rib, the fundus in the concavity of the left half of the diaphragm, the pylorus, covered by the margin of the liver, somewhat external to the right sternal line at the level of the apex of the ensiform cartilage or a little lower (individual variations). The greater curvature (lower limit of the stomach) lies several centimeters above the umbilicus.

6. The **Colon** (red outline) is readily seen in position. The cecum is situated in the right iliac fossa.

PLATE 24.

PERCUTORY AND AUSCULTATORY TOPOGRAPHY.

The most important topographic lines are: the mammillary line (M), the sternal line (St), the parasternal line (P) between the two, the median line, and the anterior, middle, and posterior axillary lines.

The clear, not tympanitic percussion-note of the lung (blue outline) extends on the right side in the mammillary line to the lower border of the sixth rib. From this point downward there is absolute liver-dulness to the costal arch. Below is the clear tympanitic note of the intestine. Upon the left side the pulmonary note in the mammillary line is only indistinctly separable from the tympanitic note of the contiguous stomach.

The area of absolute cardiac dulness (doubly hatched red lines) begins at the left sternal line, at the lower border of the left fourth rib, extending on the right to the left sternal line and on the left to a point somewhat within the mammillary line.

The area of relative cardiac dulness (portions of the heart covered by lung) becomes distinct only at times on deep percussion (horizontal red lines). Below, the cardiac dulness merges with the liver-dulness.

Traube's semilunar space (interrupted black line) yields a deeply tympanitic note from the stomach. Its boundaries are, above, the lower border of the left lung; on the right, the liver-dulness; below, the costal arch; on the left (see Plate 28, *b*), the splenic dulness.

The points for auscultation of the heart-sounds are as follows:

1. For the aorta, the right sternal margin in the second intercostal space (A), where are heard: duk **duk** duk **duk** (second sound louder than the first).

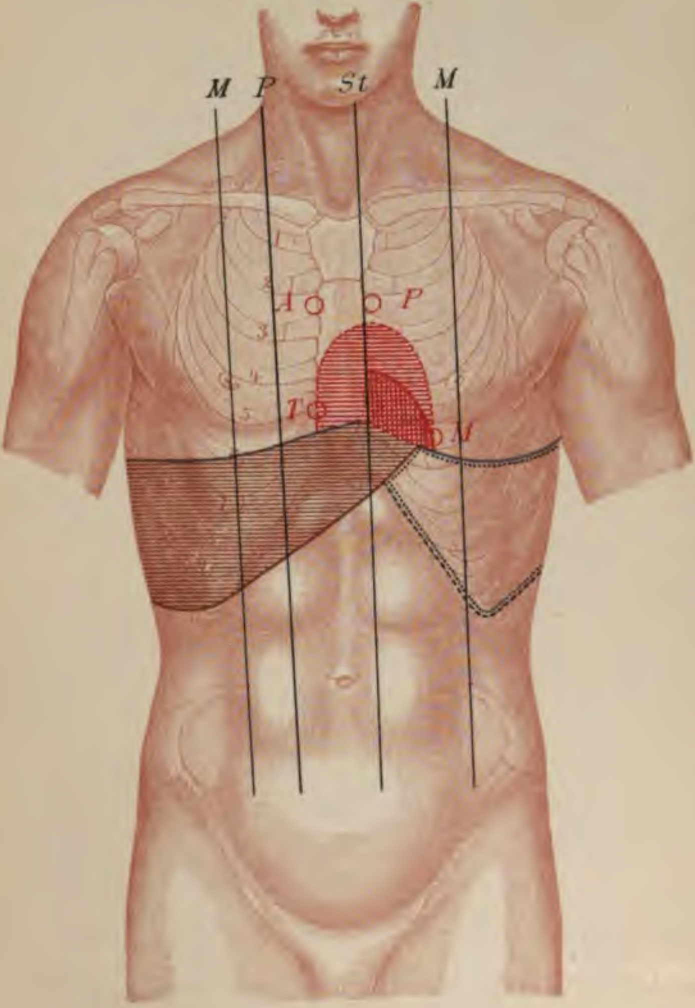
2. For the pulmonary artery, the left sternal margin in the second intercostal space (P), where are heard: duk **duk** duk **duk**.

3. For the mitral, the fifth intercostal space somewhat within the left mammillary line (M), where are heard: **duk** duk **duk** duk (systolic sound the louder).

4. For the tricuspid, insertion of fifth right rib, where are heard: **duk** duk **duk** duk.

(For the theory of heart-sounds see Epitome, Section II., Auscultation, p. 33.)

Tab. 24.



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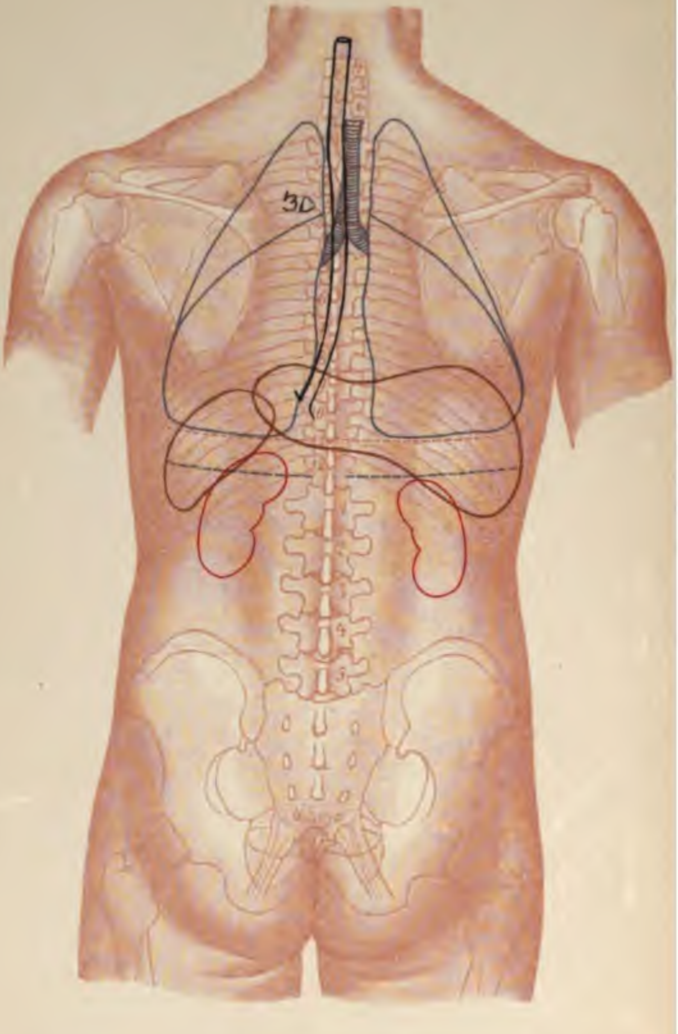

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PLATE 25.

PROJECTION OF THE INTERNAL ORGANS UPON THE POSTERIOR ASPECT OF THE BODY.

1. **Lungs** (blue outline).—The lower limits of the pleuræ (interrupted blue line) extend lower upon both sides than the lower limits of the lungs. The upper lobe of each lung extends posteriorly to the level of the spinal process of the third dorsal vertebra, and from this point downward extends the lower lobe (to the spinal process of the eleventh dorsal vertebra). The bifurcation of the trachea takes place between the third and fourth dorsal vertebræ, the right bronchus being somewhat larger than the left.

2. The **Esophagus** (black outline) pursues a slightly curved course along the spinal column. The distance from·the mouth to the intersection of the left bronchus is 23 cm. (9 in.), to the cardia, 40 cm.—15¾ in.—(in adults).

3. The **Spleen** (brown outline) extends from the ninth to the eleventh rib on the left side. Anteriorly, it does not extend beyond the costo-articular line (from the left sternoclavicular articulation to the apex of the eleventh rib). It is normally not palpable.

4. The **Liver** (brown outline) is in this illustration largely covered by lung; on the right side inferiorly and laterally it is in contact with the body-wall (in the axillary line between the tenth and eleventh ribs).

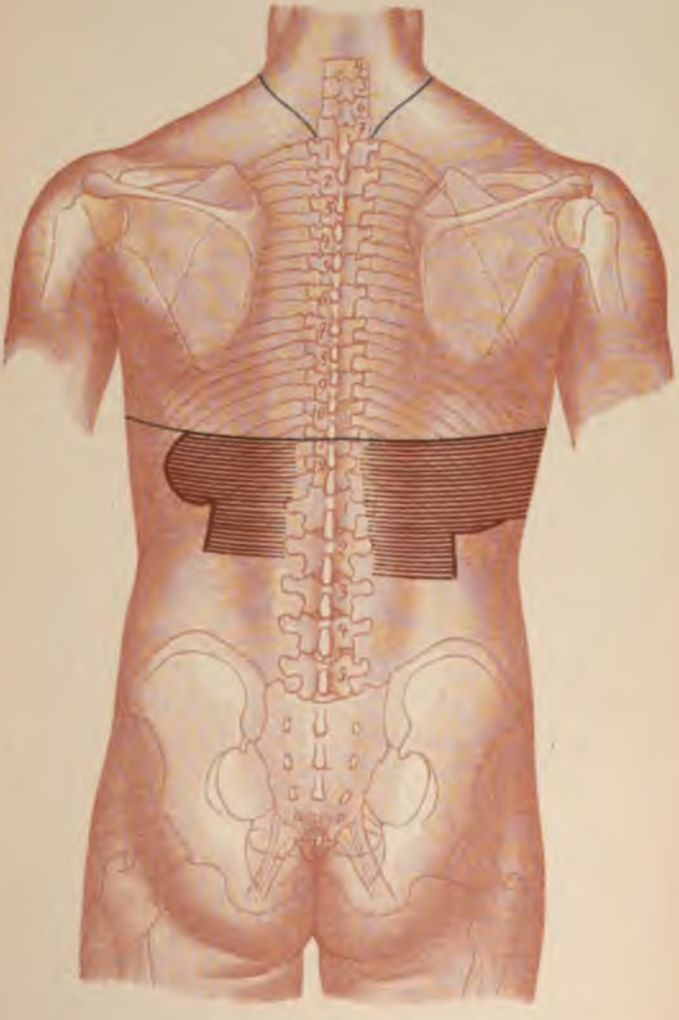
5. The **Kidneys** (red outline).—They extend from the twelfth dorsal to the upper border of the third lumbar vertebra. The left kidney is somewhat higher than the right.

PLATE 26.

PERCUTORY TOPOGRAPHY OF THE BACK.

The clear, not tympanitic pulmonary percussion-note extends on both sides from above (apex of the lung) downward (blue outline). At the level of the spinal process of the eleventh dorsal vertebra it gives way on the left to the splenic dulness and on the right to the liver-dulness (brown). Below both of these, on either side, is the kidney-dulness (red). The lower limit of the latter is at the level of the upper border of the third lumbar vertebra, the anterior limit about 10 cm. (4 in.) from the vertebral column, forming on the left with the lower limit of the splenic dulness the splenorenal angle of dulness, and on the right with the lower limit of liver-dulness the hepatorenal angle of dulness. On auscultatiôn vesicular breathing is to be heard throughout the whole area of pulmonary resonance, only over the right apex with a blowing quality (tracheal breathing).

Tab. 26.



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Tab. 27.

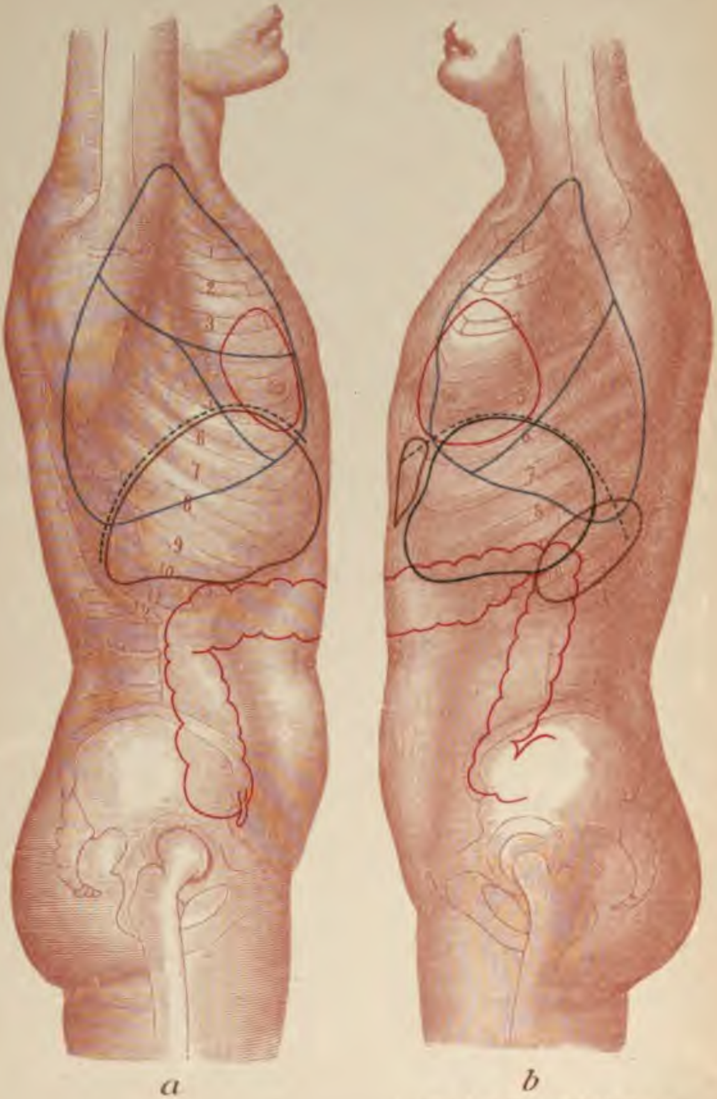

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PLATE 27.

PROJECTION OF THE INTERNAL ORGANS UPON THE LATERAL ASPECTS OF THE TRUNK.

FIG. *a*, right; FIG. *b*, left side.

To be compared with the explanation of Plate 23, which applies also to this.

Lung (blue) with its lobes.

Heart (red).

Diaphragm (interrupted black line).

Liver (brown).—Its position in the concavity of the right half of the diaphragm is readily appreciated (Fig. *a*).

Stomach (black).—The fundus is situated in the concavity of the left half of the diaphragm (Fig. *b*).

Spleen (brown).

Colon (red).

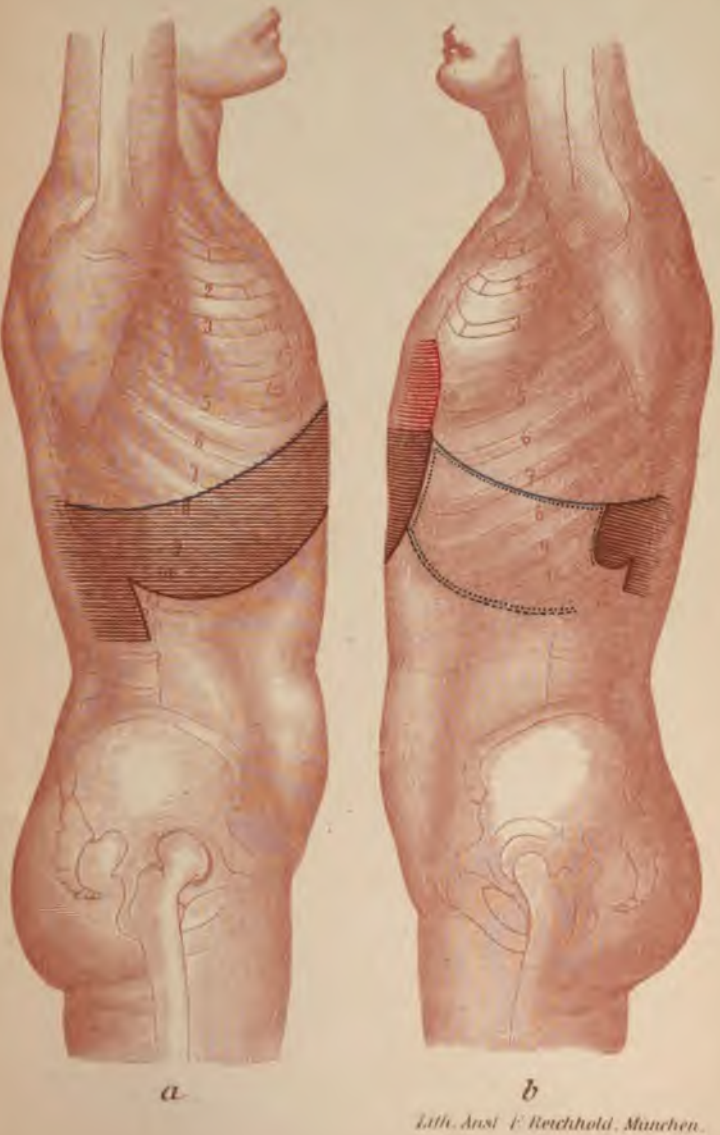
PLATE 28.

PERCUTORY TOPOGRAPHY OF THE LATERAL ASPECTS OF THE TRUNK.

FIG. *a*. Right side (compare text of Plate 24). Down to the lower limits of the lung (blue) the percussion-note is clear but not tympanitic, meeting the liver-dulness (brown). The lower limit of liver-dulness lies in the axillary line between the tenth and eleventh ribs, follows the costal arch to the mammillary line, and passes upward midway between the ensiform cartilage and the umbilicus. Over the eleventh rib is the hepatorenal angle of dulness.

FIG. *b*. Left side. Down to the blue line the pulmonary percussion-note is clear but not tympanitic; here the note (limit not sharply defined) passes into the deeply tympanitic note of Traube's space (dotted black line); the posterior limit (brown, splenic dulness) is here to be seen. (Compare explanation of Plate 24.)

Area of cardiac percussion-dulness (red); hepatic percussion-dulness (brown).



a *b*

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DIAGRAMMATIC REPRESENTATION

OF

DISEASES OF THE LUNGS.

(Plates 29–42.)

Tab. 29.

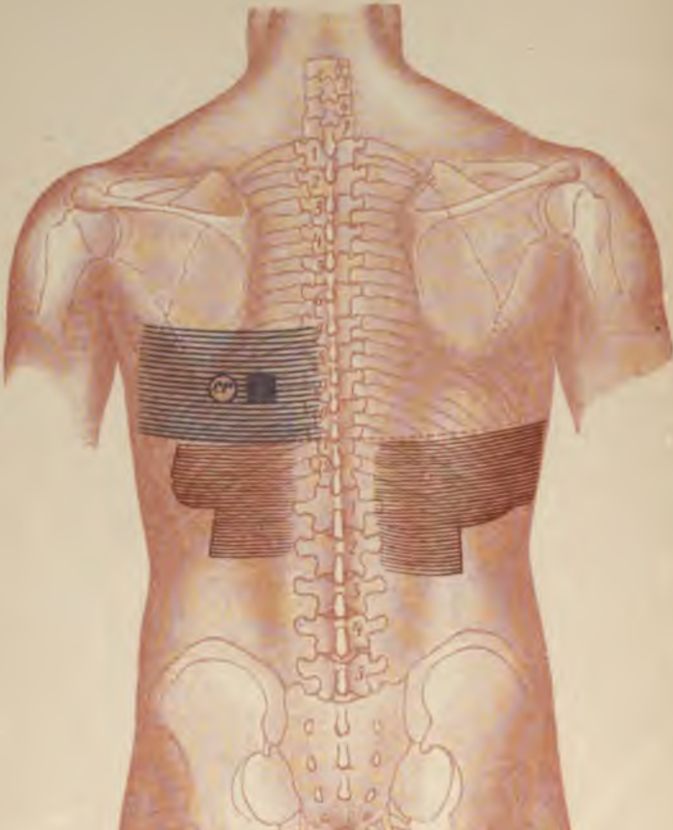

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PLATE 29.

CROUPOUS PNEUMONIA OF THE POSTERIOR INFERIOR PORTION OF THE LEFT SIDE.

History.—A soldier, 23 years old, was seized suddenly on the evening of April 24 at 8.30 o'clock, with a severe chill, vomiting, and pain in the left side of the chest. Soon there appeared irritative cough and a sense of heat. The temperature was 39.6° C. (103.3° F.). Objective examination yielded negative results.

Treatment.—Mustard-leaf, cool applications, lemonade.

On the following morning the temperature was 39.4° C. (102.9° F.), the pulse 108. The subjective symptoms were unchanged. The sputum was viscid and bloody.

Physical Examination (see plate) disclosed at the base of the left chest posteriorly a hand's-breadth area of slight dulness, with a distinct tympanitic note. On auscultation slight blowing breathing was heard, with numerous inspiratory crepitant râles. Vocal fremitus was not diminished. A leukocytosis of 25,000 existed.

Diagnosis.—Croupous pneumonia of the left lower lobe in the ~~stage of congestion~~.

Treatment.—Seltzer water with milk, lemonade with white wine. An injection of morphin (0.01—gr. $\frac{1}{6}$) was given for the relief of the severe, sharp pain in the side and an ice-bag was applied in this situation.

PLATE 30.

CROUPOUS PNEUMONIA.

The patient whose case is illustrated in Plate 29 developed continued high fever, much thirst, headache, and respiratory difficulty. On the fifth day the temperature was 40.2° C. (104.4° F.), the pulse 118. The sputum was tough and brownish-red in color and contained microscopically many diplococci. Slight labial herpes had been present for two days and there were marked dyspnea and cyanosis. The leukocytosis had reached 42,000.

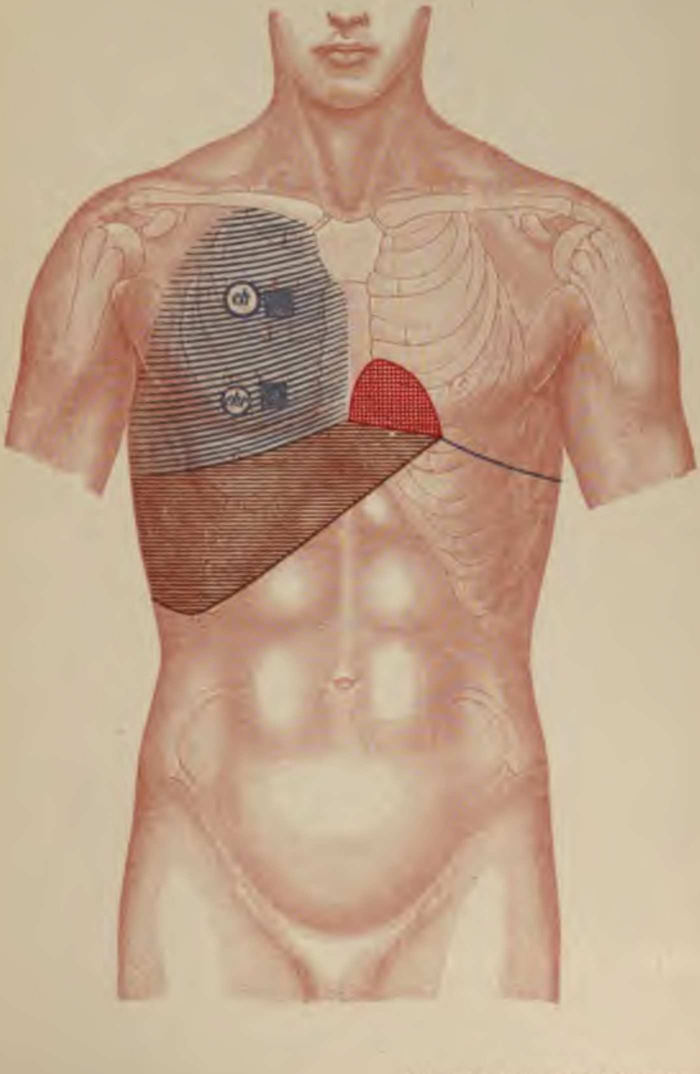
On **Percussion** over the upper portion of the left half of the chest the note was tympanitic (relaxation of the apex of the lung). ① From the second intercostal space downward there was dulness of progressive intensity, with a tympanitic accompaniment. The dulness passed over into that of the liver, the lower border of which was not displaced. On auscultation vesicular breathing was audible over the apex of the lung, without adventitious sounds. Over the second and third intercostal spaces was heard loud bronchial breathing, without adventitious sounds. Over the fourth and fifth intercostal spaces was heard bronchial breathing, with numerous fine mucous râles (especially on inspiration).

Diagnosis.—Croupous pneumonia of the lower half of the left upper lobe, in the stage of hepatization, in the middle in the stage of absorption.

Treatment.—Equal parts of powdered digitalis and camphor (0.1—gr. jss); cold applications. At night an injection of morphin (0.015—gr. ¼) was given.

① Compensatory incr. air.

Tab. 30.



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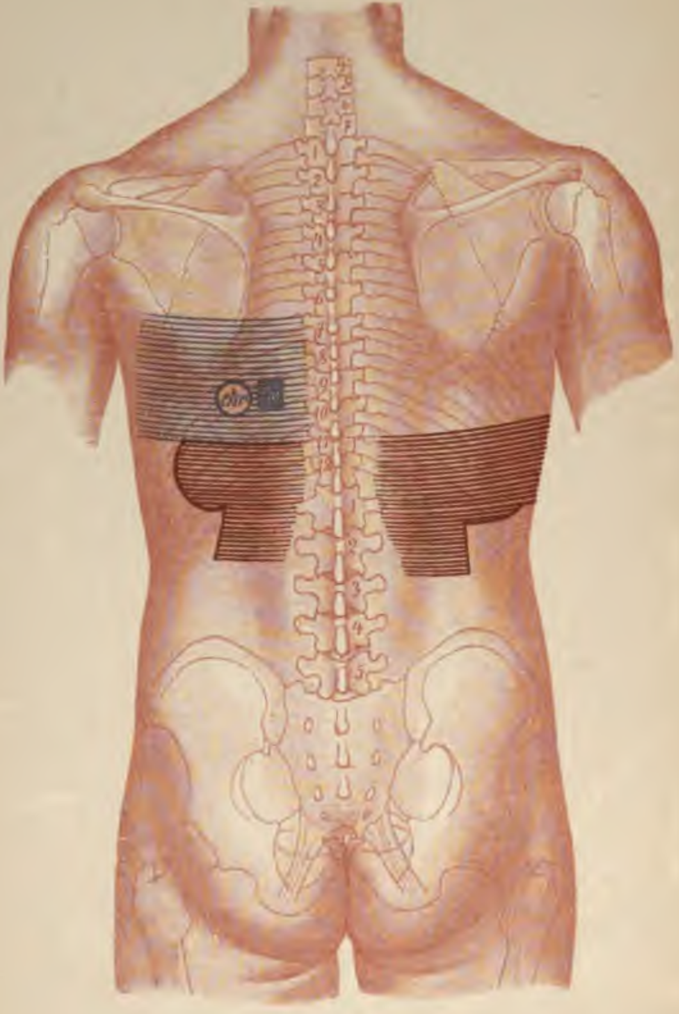

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PLATE 31.

EXUDATIVE PLEURISY ON THE LEFT SIDE POSTERIORLY.

History.—A woman, 30 years old, whose father and one sister had died of tuberculosis, had not felt well for some time and complained of irritative cough, pain in the side of the chest, evening chilliness, and loss of appetite. There was no expectoration.

On **Examination** the temperature was found to be 38.4° C. (101.1° F.), the pulse 110.

On **Percussion** a hand's-breath area of intense dulness, with a sense of marked resistance, was found upon the posterior aspect of the lower portion of the left half of the chest, beginning at the level of the spinous process of the seventh dorsal vertebra.

On **Auscultation** feeble blowing breathing was heard, with fine crepitant râles during inspiration. Vocal fremitus was markedly diminished.

Exploratory puncture disclosed the presence of a serous exudate, which upon inoculation and culture proved sterile.

Diagnosis.—Exudative pleurisy (tuberculous?) of the left side.

Treatment.—Diuretics. Cold packs. Ems water with milk. Sustaining diet.

PLATE 32.

EXUDATIVE PLEURISY ON THE LEFT SIDE.

The patient whose case is illustrated in Plate 31 exhibited on the following days slowly ascending, irregular fever, profuse sweats (especially at night), increasing respiratory difficulty, and palpitation of the heart. Sleep and appetite were poor.

Examination on the seventh day disclosed a temperature of 39.1° C. (102.4° F.), and a pulse of 126, with considerable dyspnea and slight cyanosis.

On **Percussion** a highly tympanitic, ringing note was elicited at the upper portion of the anterior aspect of the left half of the chest, as low as the second intercostal space. From this point downward there was intense dulness passing into that of the cardiac area (oblique upper limit). Traube's semilunar space exhibited total dulness in its upper half, while in its lower half the note was highly tympanitic and ringing (gastric note). The right limit of absolute cardiac dulness extended beyond the right margin of the sternum.

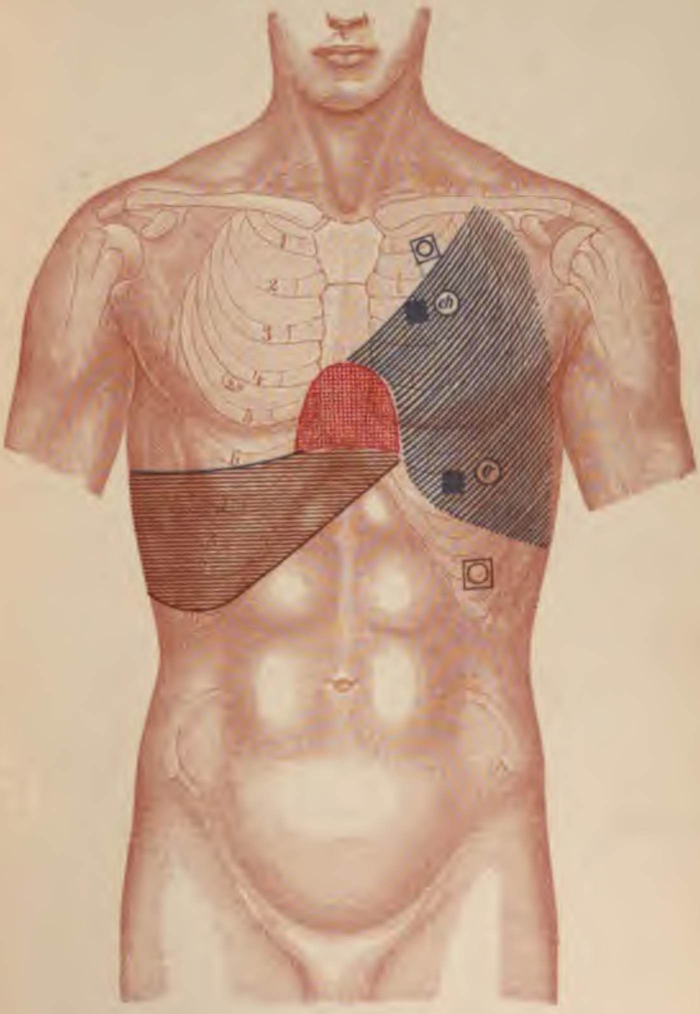
On **Auscultation** the breath-sounds were wanting over the lower half of the area of dulness and greatly enfeebled over the upper half, while the distant bronchial breathing was attended with a small number of fine, moist râles.

The heart-sounds were normal.

Diagnosis.—A large pleuritic effusion, filling up Traube's space and displacing the heart.

Treatment consisted in puncture. The needle was introduced posteriorly (where the exudate reached the level of the spinal process of the fourth dorsal vertebra) in the eighth intercostal space, and somewhat more than 1000 c.c. (a quart) of serous fluid was evacuated.

Tab. 32.



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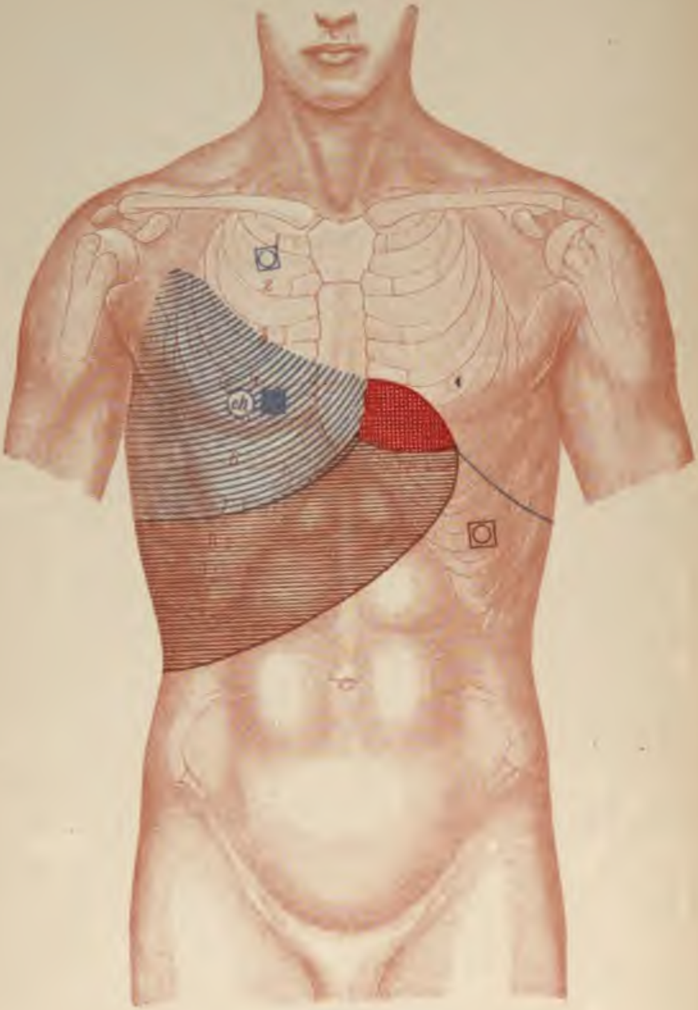

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PLATE 33.

EXUDATIVE PLEURISY ON THE RIGHT SIDE.

History.—A student, 24 years old, had undertaken a short journey and found that his breathing was interfered with. In other respects he felt quite well [?].

On **Examination** the temperature in the morning was found to be normal (the pulse 96). Dyspnea was apparently not pronounced.

On **Percussion** the note was found clear and slightly tympanitic at the upper portion of the anterior aspect of the right half of the chest. From the upper border of the third rib downward there was profound dulness, with a sense of resistance, merging with the area of hepatic percussion-dulness. The left limit of cardiac dulness extended to the mammillary line, where the apex-beat also was visible. The area of hepatic percussion-dulness extended two fingers' breadth below the costal margin. Posteriorly dulness on percussion extended from the spinous process of the fifth dorsal vertebra downward, with abolition of vocal fremitus.

On **Auscultation** there was scarcely audible blowing breathing over the area of dulness, without adventitious sounds.

Diagnosis.—Exudative right-sided pleurisy, with displacement of heart and liver.

Treatment consisted in puncture, 900 cu. cm. of serous exudate being evacuated. Recovery was uncomplicated.

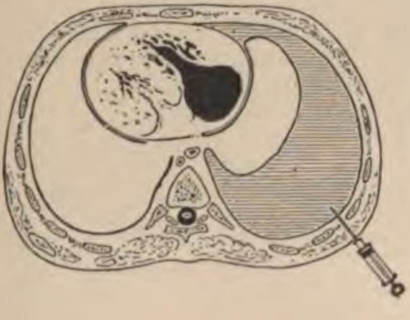


PLATE 34.

HYDROTHORAX.

History.—A young man of 18 years had felt ill for two weeks. There had been frequent vomiting, with loss of appetite, respiratory difficulty, irritative cough, and swelling of the lower extremities.

On **Examination** the temperature was found normal, the pulse 80. There was edema of the eyelids, the cheeks, and the extremities.

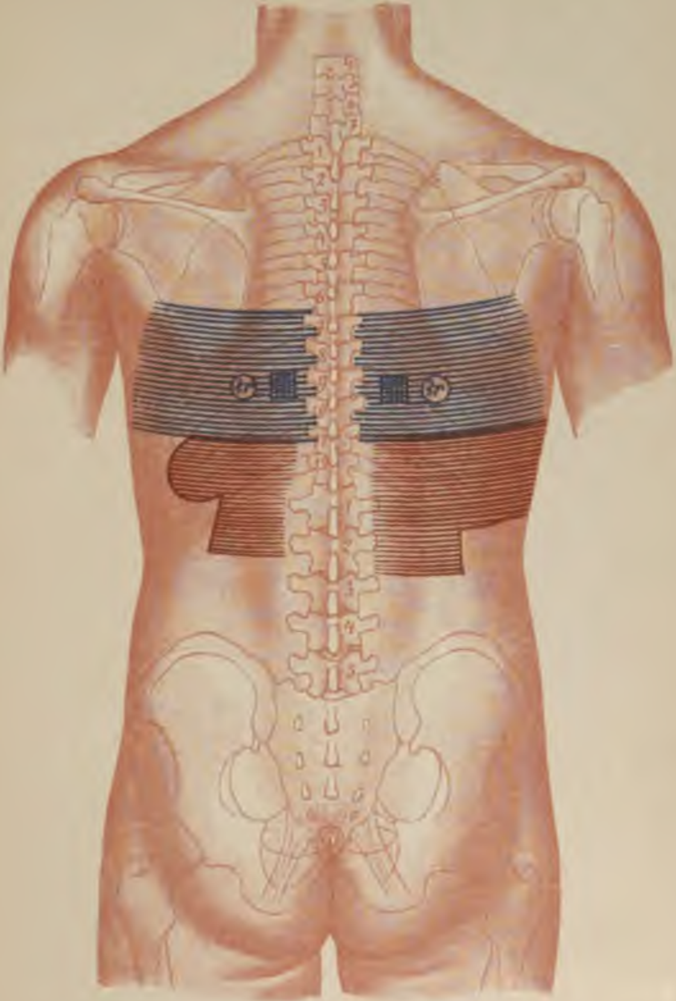
On **Percussion** there was found marked dulness on both sides posteriorly, beginning at the level of the spinal process of the fifth dorsal vertebra, and progressively increasing in intensity downward. Inferiorly there was a pronounced sense of resistance.

On **Auscultation** there was found throughout the areas of dulness on both sides enfeebled vesicular breathing, with numerous crepitant râles during inspiration, and diminished vocal fremitus. The urine contained a considerable amount of albumin, etc.

Diagnosis.—Acute nephritis, with edema and hydrothorax.

Treatment.—Diaphoresis, warm baths, warm packs, with milk-diet and later mild diuretics.

Tab. 34.



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Tab. 35.

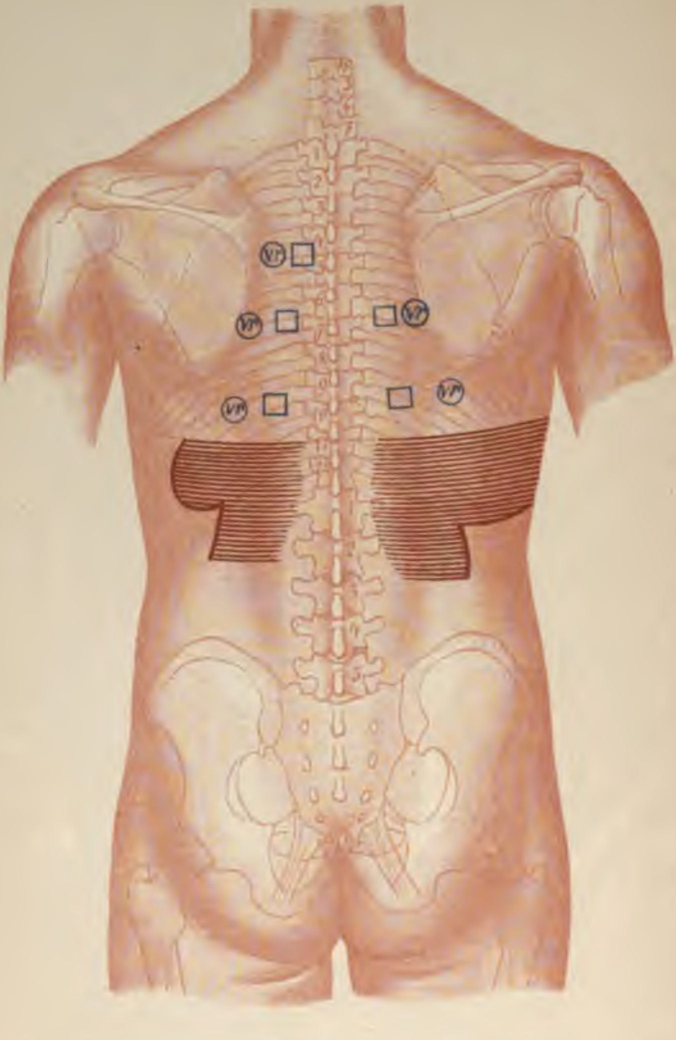

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PLATE 35.

ACUTE DIFFUSE BRONCHITIS.

History.—A woman, 35 years old, was seized rather suddenly with chilliness, cough, and hoarseness. The irritative cough grew more and more distressing, while the expectoration was tough and viscid. There were also respiratory difficulty, languor, and loss of appetite.

On **Examination** the temperature was found to be normal, the pulse 80. The sputum was mucopurulent.

On **Percussion** the limits of pulmonary resonance were found normal, and everywhere the note was clear and resonant.

On **Auscultation** there were found posteriorly upon both sides, but especially over the lower portions, roughened vesicular breathing, with somewhat prolonged expiration, accompanied by many medium-sized and fine mucous râles, intermingled, especially above, with coarse, snoring, and sibilant râles. This snoring was distinctly appreciable to the applied hand. The physical conditions were the same anteriorly, although the râles were less numerous.

Diagnosis.—Acute diffuse bronchitis.

Treatment.—Alkaline waters, tea, expectorants, codein (0.02—gr. ⅓).

PLATE 36.

BRONCHO-PNEUMONIA.

History.—In a case of typhoid fever there occurred at the end of the second week, in conjunction with cough that had been present from the beginning, an increase in the respiratory difficulty and in the expectoration, with much irritative cough and catarrhal sputum of a purulent but not bloody character.

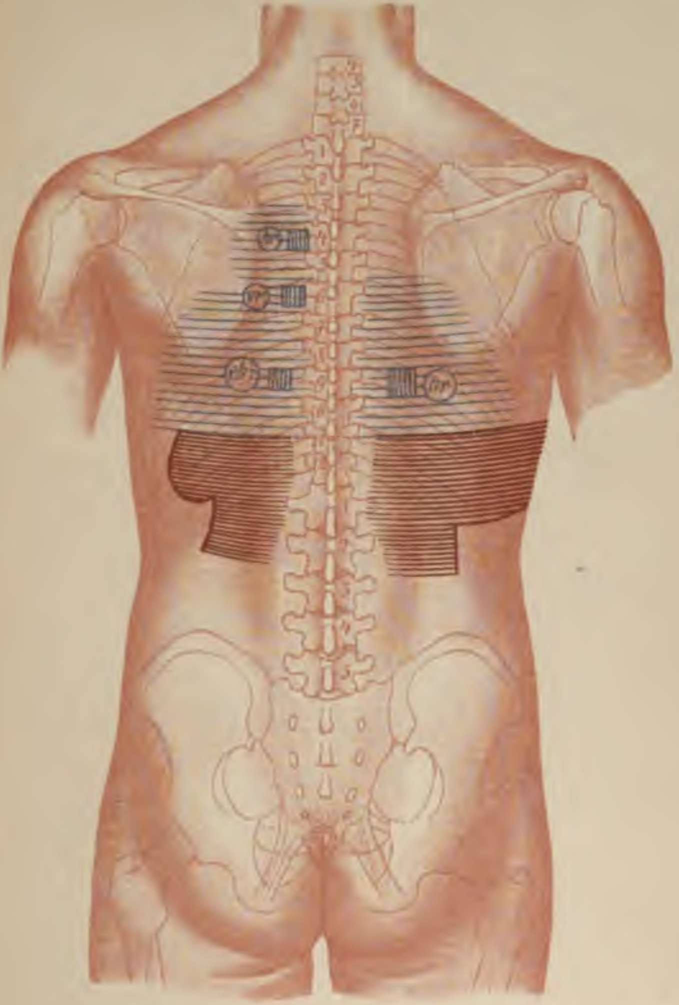
On **Examination** the temperature in the evening was found to be 40.5° C. (104.9° F.), the pulse 128.

On **Percussion** there was found over the lower portion of both lungs a hand's-breadth area of not very pronounced, yet distinct, dulness (more marked upon the left than upon the right), with a distinctly tympanitic note on the left.

On **Auscultation** the breath-sounds were found at times almost entirely wanting over the area of dulness (accumulation of secretion), but after efforts at cough distinct bronchial breathing was audible (upon the right of a more pronounced blowing character). The breathing was almost entirely obscured by the very abundant moist, medium-sized, and coarse mucous râles.

Diagnosis.—Broncho-pneumonia complicating typhoid fever.

Treatment.—Cold affusions in the bath, with moist applications and expectorants.



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Tab. 37.

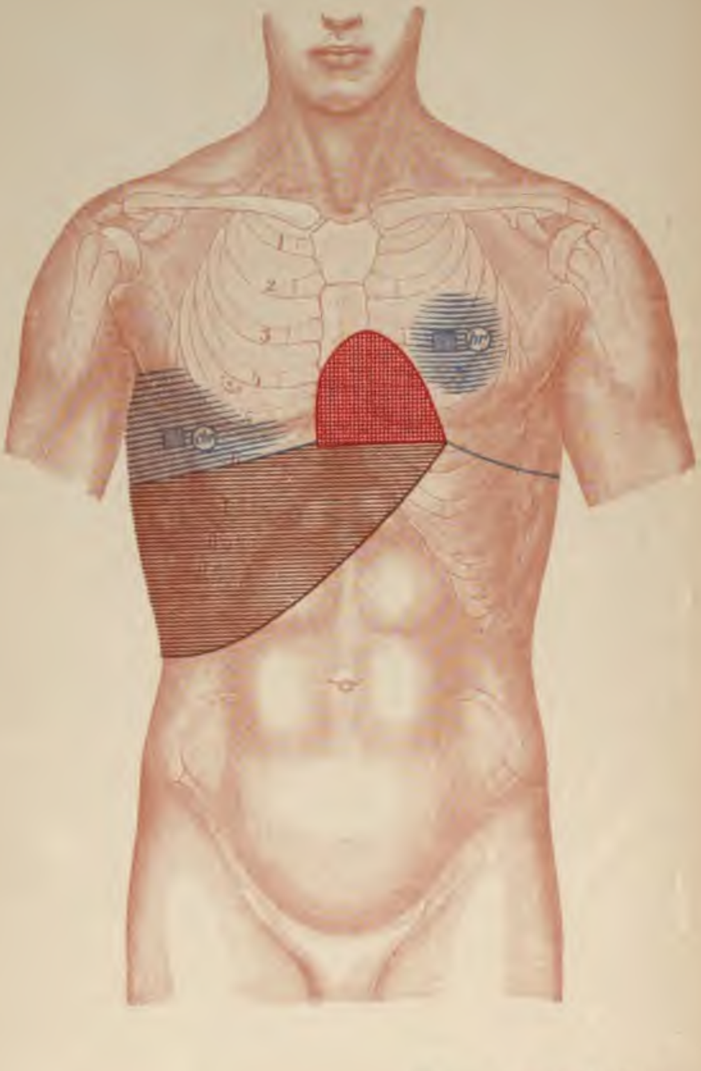

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PLATE 37.

PULMONARY INFARCTION.

History.—In the case of a man suffering from a grave cardiac lesion (mitral insufficiency and stenosis) there set in suddenly extreme dyspnea and distressing irritative cough. After a short time bloody sputum was ejected and there was severe pain in the side of the chest.

On **Examination** the temperature was found to be 38.7° C. (101.7° F.), the pulse 136. The sputum consisted of almost pure blood and was liquid.

On **Percussion** there was found over the upper portion of the anterior aspect of the left chest, and especially laterally, an area of slightly impaired resonance of ill-defined limitation. A similar area with more pronounced impairment of resonance was present upon the right side, laterally and posteriorly.

On **Auscultation** there was heard over both areas indistinct vesicular breathing (in a circumscribed area further posteriorly on the right there was distinct blowing breathing), with numerous moist râles.

Diagnosis.—Multiple hemorrhagic infarction of the lung.

Treatment.—Powdered digitalis (0.01—gr. $\frac{1}{6}$) every two hours, with morphin and the application of an ice-bag.

PLATE 38.

PULMONARY EMPHYSEMA.

History.—A workman, 56 years old, employed in a textile mill, had suffered for years with cough and respiratory difficulty. For a week there had been, as often before, an aggravation of the symptoms.

On **Examination** the temperature was found to be normal, the pulse 68. The breathing was laborious and stridulous, and participated in by the auxiliary muscles of respiration. The sputum was abundant and purulent. The thorax was barrel-shaped.

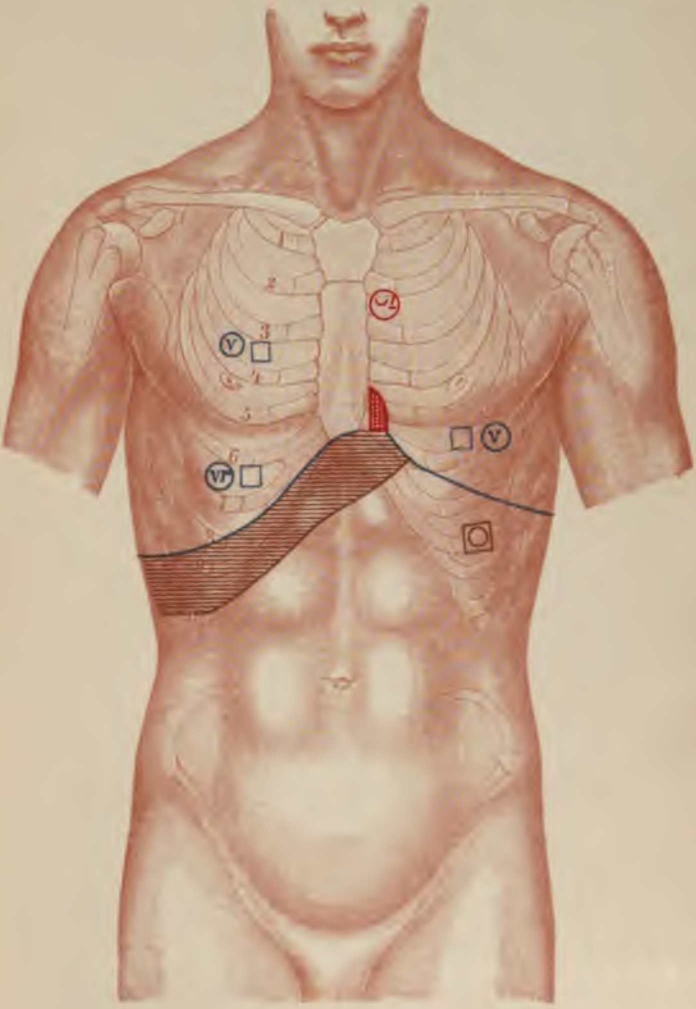
On **Percussion** the note upon both sides was found to be clear and full, though not tympanitic; upon the right, in the mammillary line, down to the lower border of the eighth rib, at which point liver-dulness began; upon the left, in the sternal line, down to the lower border of the fifth rib. The cardiac percussion-dulness was reduced to a small area at the sternal margin in consequence of overlapping of the lungs. Distinct displacement of the lower limit of the right lung upon deepest inspiration was not demonstrable.

On **Auscultation** there was heard on both sides rough vesicular breathing, with excessively prolonged expiration, and abundant sonorous and groaning râles. The pulmonary second sound was markedly accentuated, from hypertrophy of the right ventricle.

Diagnosis.—Pulmonary emphysema with rarefaction of the pulmonary structure (not merely increased volume) and chronic bronchitis.

Treatment.—Rest, expectorants, potassium iodid, morphin.

Tab. 38.



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Tab. 39.

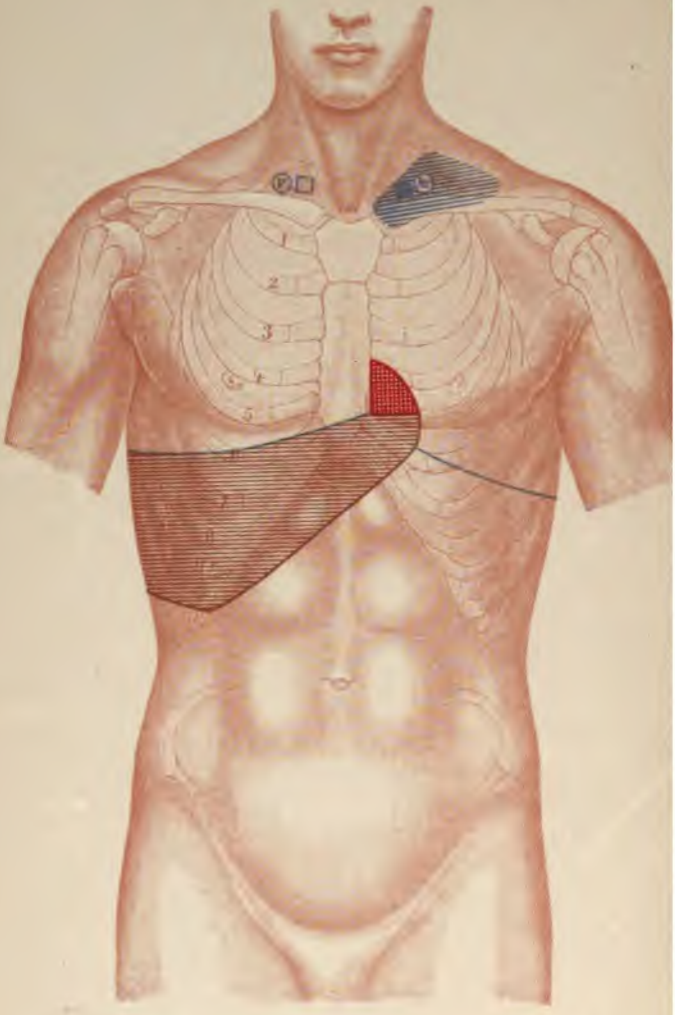

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PLATE 39.

INCIPIENT PULMONARY TUBERCULOSIS.

History.—A girl, 16 years old, with hereditary predisposition (father dead of pulmonary disease), manifested slowly progressive emaciation, cough and expectoration, with pallor and fatigue.

On **Examination** the temperature in the morning was found to be normal, the pulse 108. Chlorosis was marked and venous blowing in the ears was present. The sputum was purulent. The thorax was small, flat and long, and emaciated. The left supraclavicular fossa was somewhat retracted.

On **Percussion** moderately marked impairment of resonance was found over the apex of the left lung. Upon the right the percussion-note was clear.

On **Auscultation** the breathing at the left apex was enfeebled and ill-defined, with prolonged blowing expiration and numerous fine moist râles. The sputum contained tubercle-bacilli.

Diagnosis.—Tuberculosis of the apex of the ~~right~~ L. lung.

Treatment.—Milk-diet, rest in bed, pills of iron and arsenic later residence in the country.

PLATE 40.

PROGRESSIVE PULMONARY TUBERCULOSIS.

History.—A laborer, 25 years old, presented cough for a year and a half, had been bed-ridden for two months, was greatly emaciated, sweated profusely especially at night, had been almost completely hoarse for three months, and had had diarrhea for several weeks. Expectoration was copious. During the night there was sudden evacuation of in the neighborhood of eight ounces of bright-red blood.

On **Examination** the temperature was found to be 39.2° C. (102.6° F.), the pulse 116. The sputum consisted of almost pure blood, partly liquid, partly clotted.

On **Percussion**, made with great care, dulness was found upon the anterior aspect of the right chest from above down to the fourth intercostal space. From this point the note was clearer to the upper margin of the seventh rib. In the second intercostal space ~~tympany~~ was marked, and became more pronounced and more distinct when the mouth was opened (Wintrich's change of note). There was also dulness on percussion over the apex of the left lung anteriorly.

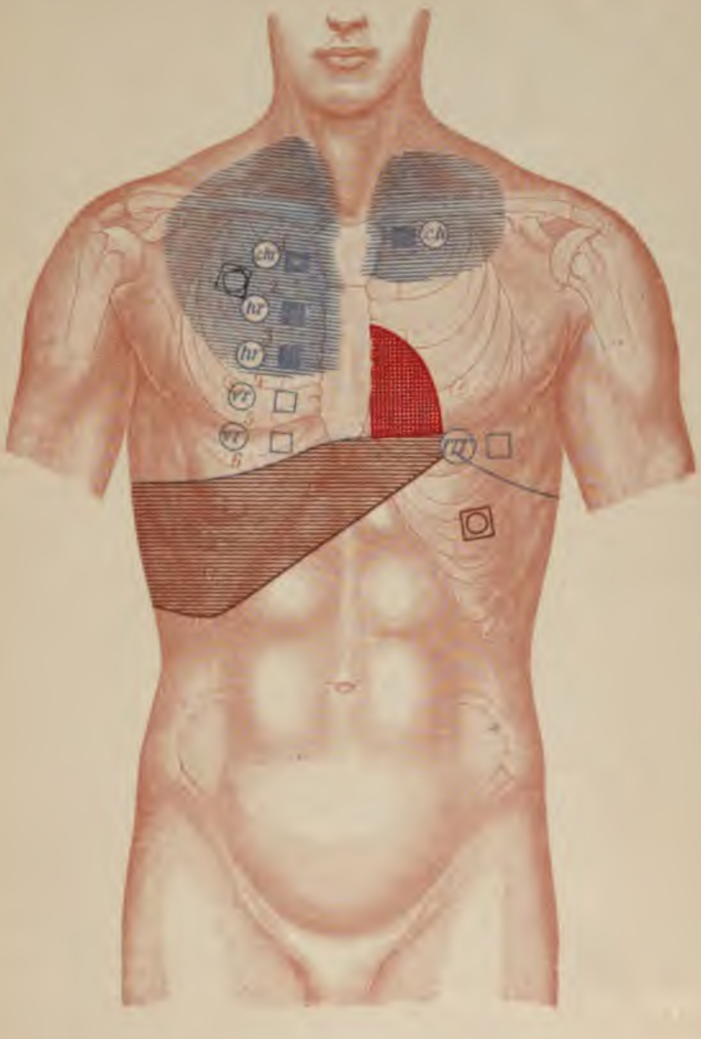
On **Auscultation** the amphoric **bronchial breathing** at the upper portion of the anterior aspect of the right chest was at times entirely obscured by numerous coarse, moist crackling and sonorous râles; lower down the breathing was loud and bronchial, with medium-sized moist râles. Over the upper portion of the anterior aspect of the left chest the breathing was blowing, with isolated crackling.

Laryngoscopic examination disclosed ulcerative destruction of the right vocal band. (See Epitome, Fig. 37.)

Diagnosis.—Progressive bilateral pulmonary tuberculosis, with cavity-formation in the right upper lobe; laryngeal and intestinal tuberculosis; recent hemoptysis.

Treatment.—Ice-bag to the upper portion of the anterior aspect of the right chest, lemonade, pills of ergotin, morphin.

Tab. 40.



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Tab. 41.

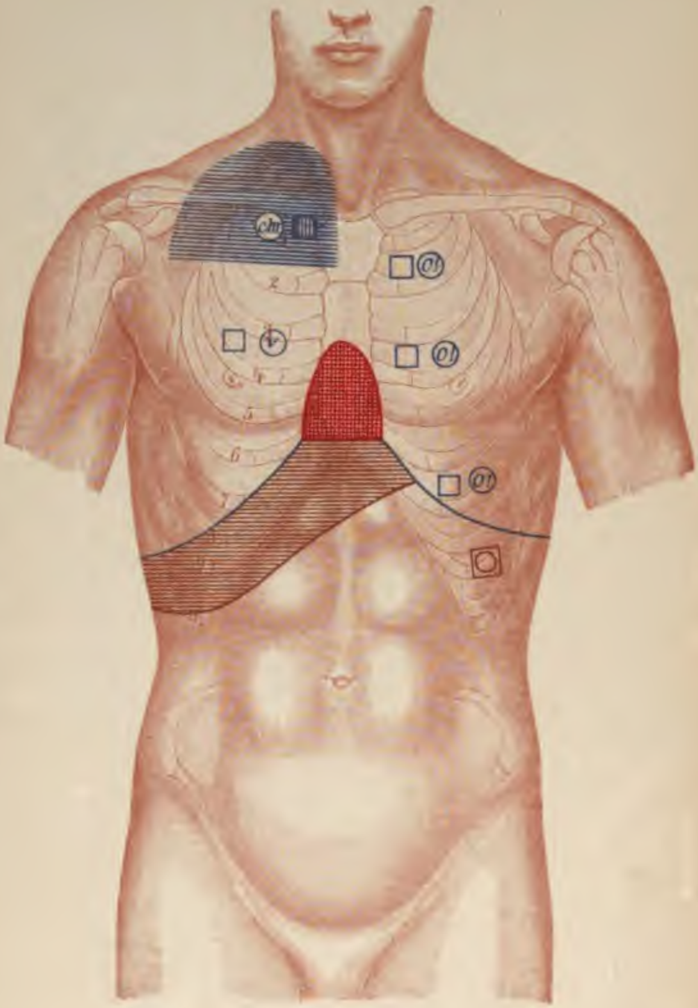

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PLATE 41.

PNEUMOTHORAX.

History.—A tuberculous subject, with physical signs of involvement of both lungs, was awakened suddenly during the night with severe dyspnea, oppression in breathing, and pains in the chest.

On **Examination** there were found to be marked dyspnea and cyanosis. The temperature was 36.4° C. (97.6° F.). The left half of the thorax was greatly distended and took no part in respiration.

On **Percussion** there was found upon both sides anteriorly clear, not tympanitic, pulmonary resonance, which appeared only upon the left to be strikingly full and deep. Upon the right side the clear note extended to the lower border of the seventh rib (vicarious emphysema); upon the left the loud, not (!) tympanitic note extended down to Traube's space. The area of cardiac percussion-dulness had disappeared from its normal situation and was found over the lower portion of the sternum. The cardiac apex-beat could not be felt.

On **Auscultation** there was found, upon the right, bronchial breathing, with isolated râles, especially at the apex; below, the breathing was vesicular. Upon the left no breath-sounds at all could be heard. On percussion with the rod-pleximeter a distinct metallic note was audible. There was no succussion-sound, but occasionally metallic tinkling was heard.

Diagnosis.—Closed pneumothorax.

Treatment.—Injection of morphin.

PLATE 42.

PYOPNEUMOTHORAX.

History.—In a case of pulmonary tuberculosis the right half of the chest became strikingly distended and failed to take part in the dyspneic breathing. There had, however, been no complaint of striking change in the subjective condition during the preceding weeks.

On **Percussion** the note upon the anterior aspect of the right half of the chest was found strikingly loud, but not tympanitic, down to the fourth intercostal space, while below this point there was absolute dulness. The area of hepatic percussion-dulness extended three-fingers' breadth beyond the costal margin.

Over the anterior aspect of the upper portion of the left side of the chest there was distinct dulness on percussion, with tympanitic accompaniment, down to the second intercostal space. From this point downward there was clear, not tympanitic pulmonary resonance.

On **Auscultation** the breath-sounds were wanting over the anterior aspect of the right half of the chest, but abundant metallic tinkling was present, with a metallic note on percussion with the rod-pleximeter. When the patient was shaken at the shoulders loud metallic splashing was heard throughout the whole room (succussion).

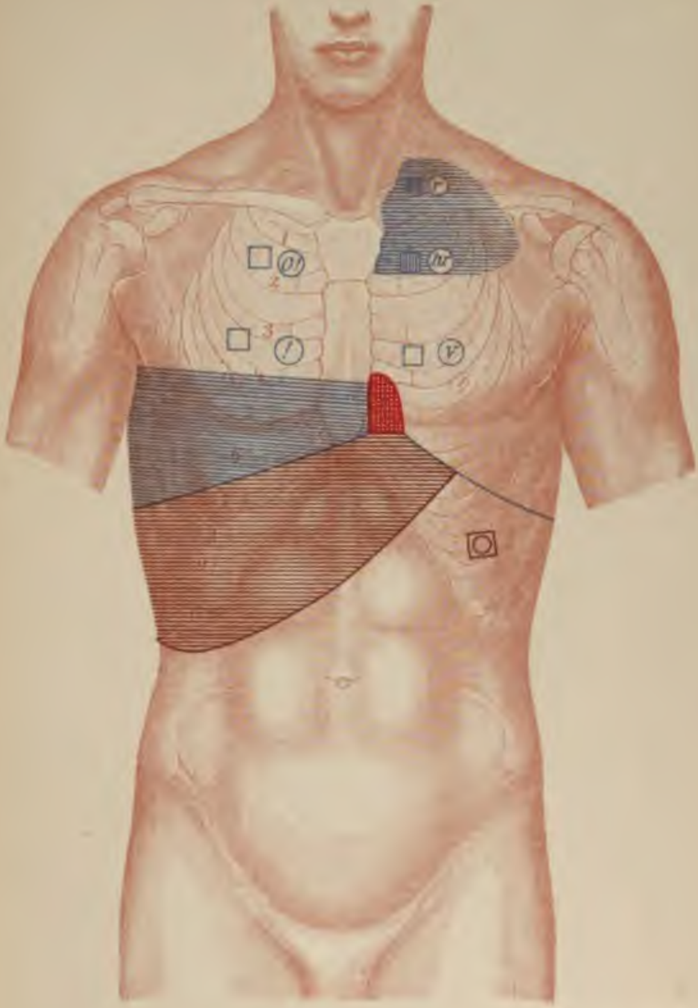
The level of the lower limit of dulness varied at once with change in the patient's posture, being higher in the erect and lower in the recumbent posture.

Exploratory puncture in the fifth intercostal space disclosed the presence of diffluent pus.

Diagnosis.—Pyopneumothorax.

Treatment.—Operative evacuation of the purulent accumulation.

Tab. 42.



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DIAGRAMMATIC REPRESENTATION

OF

DISEASES OF THE HEART.

(PLATES 43–50.)

PLATE 42.

PYOPNEUMOTHORAX.

History.—In a case of pulmonary tuberculosis the right half of the chest became strikingly distended and failed to take part in the dyspneic breathing. There had, however, been no complaint of striking change in the subjective condition during the preceding weeks.

On **Percussion** the note upon the anterior aspect of the right half of the chest was found strikingly loud, but not tympanitic, down to the fourth intercostal space, while below this point there was absolute dulness. The area of hepatic percussion-dulness extended three-fingers' breadth beyond the costal margin.

Over the anterior aspect of the upper portion of the left side of the chest there was distinct dulness on percussion, with tympanitic accompaniment, down to the second intercostal space. From this point downward there was clear, not tympanitic pulmonary resonance.

On **Auscultation** the breath-sounds were wanting over the anterior aspect of the right half of the chest, but abundant metallic tinkling was present, with a metallic note on percussion with the rod-pleximeter. When the patient was shaken at the shoulders loud metallic splashing was heard throughout the whole room (succussion).

The level of the lower limit of dulness varied at once with change in the patient's posture, being higher in the erect and lower in the recumbent posture.

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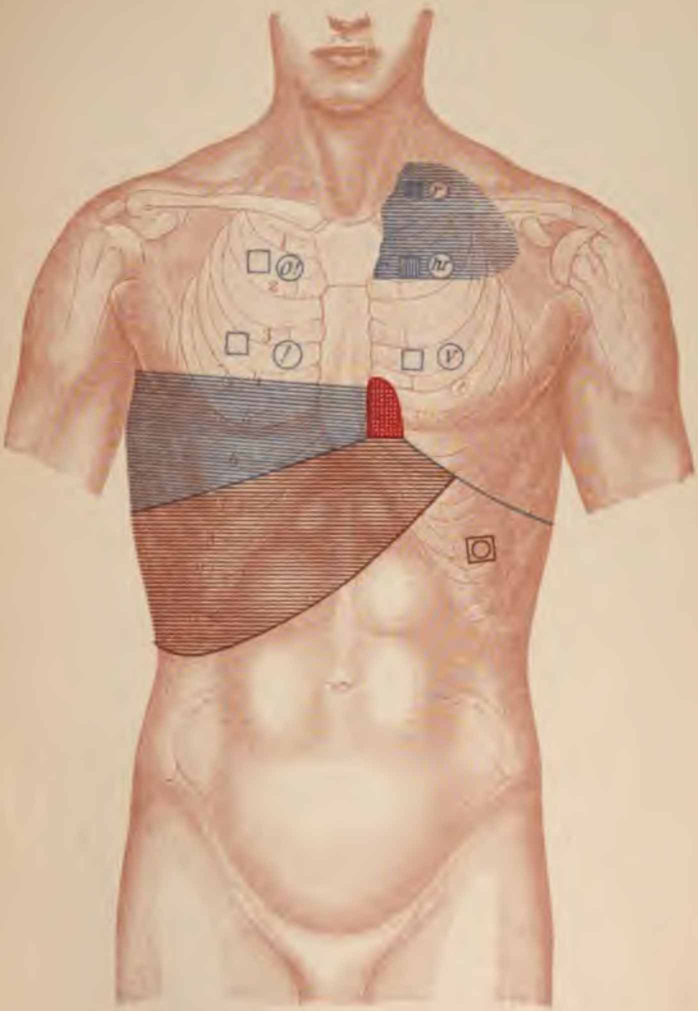

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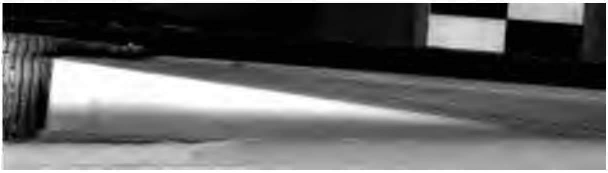

DIAGRAMMATIC REPRESENTATION

OF

DISEASES OF THE HEART.

(Plates 43-50.)

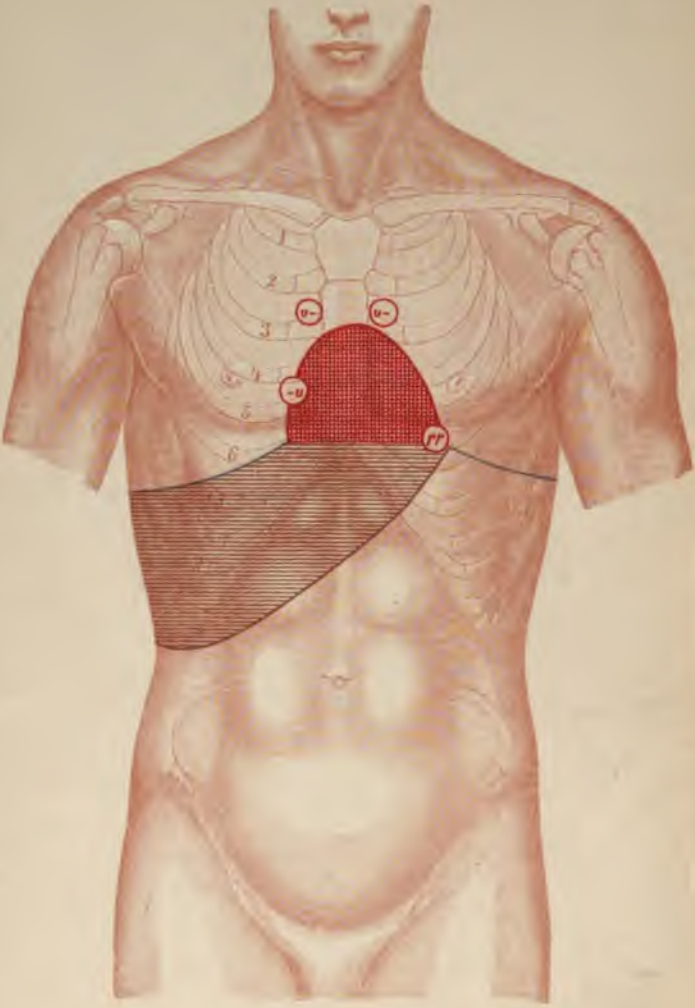

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PLATE 43.

UNCOMPENSATED CARDIAC LESION.

History.—A woman, 32 years old, had had repeatedly severe attacks of acute articular rheumatism, and had complained for years of respiratory difficulty (especially in ascending stairs) and of cough. Within three weeks these symptoms had become so aggravated that the patient could no longer go about, and, seated in bed, with the windows open, she scarcely got enough air. The lower extremities and the abdomen had been greatly swollen for two weeks and there was loss of appetite, with headache.

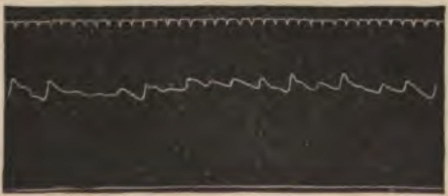
On **Examination** there were noted marked cyanosis and dyspnea (orthopnea). The temperature was normal, and the pulse (see tracing) extremely small, quite irregular, and running (not to be counted). Edema and ascites were present.

On **Percussion** the lungs were found free from noteworthy alteration (bronchitis). The upper limit of cardiac dulness was found at the lower border of the third rib; the right limit three fingers' breadth to the right of the right sternal margin; the left in the mammillary line. The apex-beat also was in the mammillary line, and there was marked epigastric pulsation. The area of hepatic dulness extended beyond the costal margin (cyanotic liver). The external jugular vein exhibited marked pulsation.

On **Auscultation** there were heard over all of the cardiac orifices two not very loud blowing murmurs (ch ch ch ch). The action of the heart was greatly accelerated and irregular, and following every four or five quick beats there were one or two slower ones.

Diagnosis.—Valvular lesion of the heart in the stage of ruptured compensation (precise diagnosis at present not possible with certainty; mitral stenosis suspected). Dilatation of the right ventricle.

Treatment.—Powdered digitalis leaves 0.15 (gr. ijss) every two hours.



Pulse-tracing of uncompensated mitral stenosis.

PLATE 44.

COMPENSATED MITRAL STENOSIS.

History.—The case represented in Plate 43, exhibited on the fourth day, after taking 2.5 grams (gr. xl) of digitalis the following phenomena:

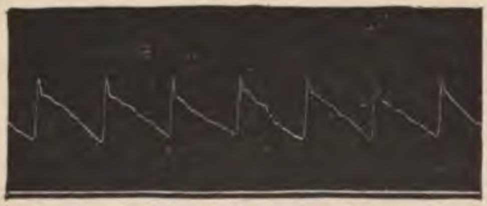
On **Examination** the temperature was found to be normal, the pulse (see tracing) 96, and almost perfectly regular and quite vigorous, though small. The edema had disappeared. Even in the recumbent posture quiet respiration was possible.

On **Percussion** the upper limit of cardiac dulness was found at the lower border of the fourth rib; the right limit slightly to the right of the median line; the left limit somewhat within the mammillary line. The area of hepatic dulness was again normal.

On **Auscultation** over the mitral orifice there was heard a presystolic (also diastolic!) short, rolling murmur preceding the first sound, with a more distinct second sound (**rduk**—duk 2° soun **rduk**—duk); over the tricuspid orifice the sounds were clear (**duk** duk **duk**); over the aorta, duk **duk** duk **duk**; over the pulmonary artery, duk **duk** (!) duk **duk** (!); the pulmonary second sound was markedly accentuated.

Diagnosis.—Stenosis of the mitral orifice; hypertrophy of the right ventricle.

(For the theory of valvular lesions, see Epitome, Section III, circulatory apparatus p. 55.)



Tracing from the same case as Plate 43, after the action of digitalis.

— = accented, or loud
= softer

Tab. 44.

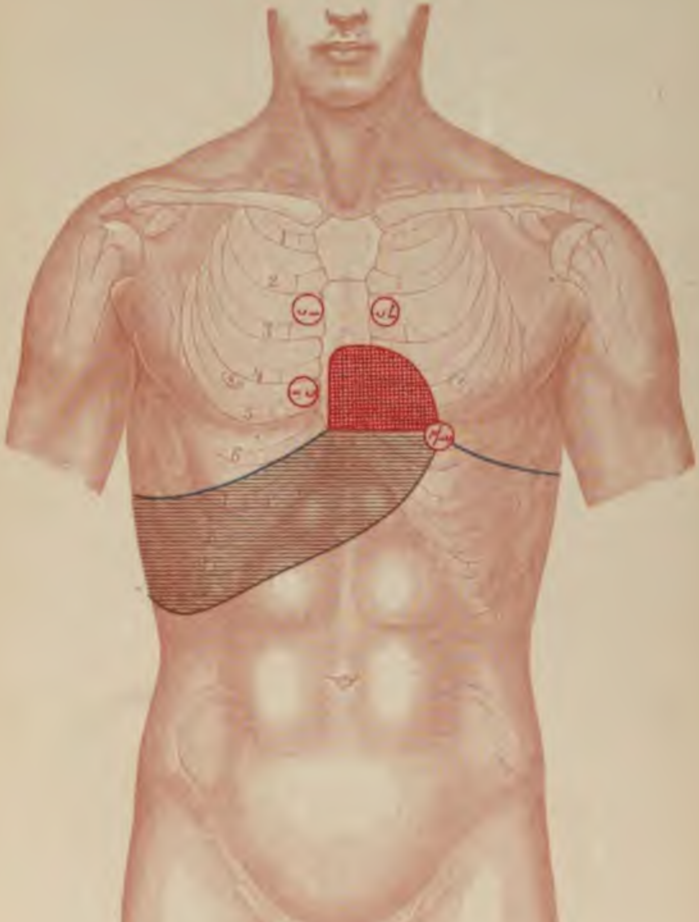


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PLATE 46.

AORTIC INSUFFICIENCY.

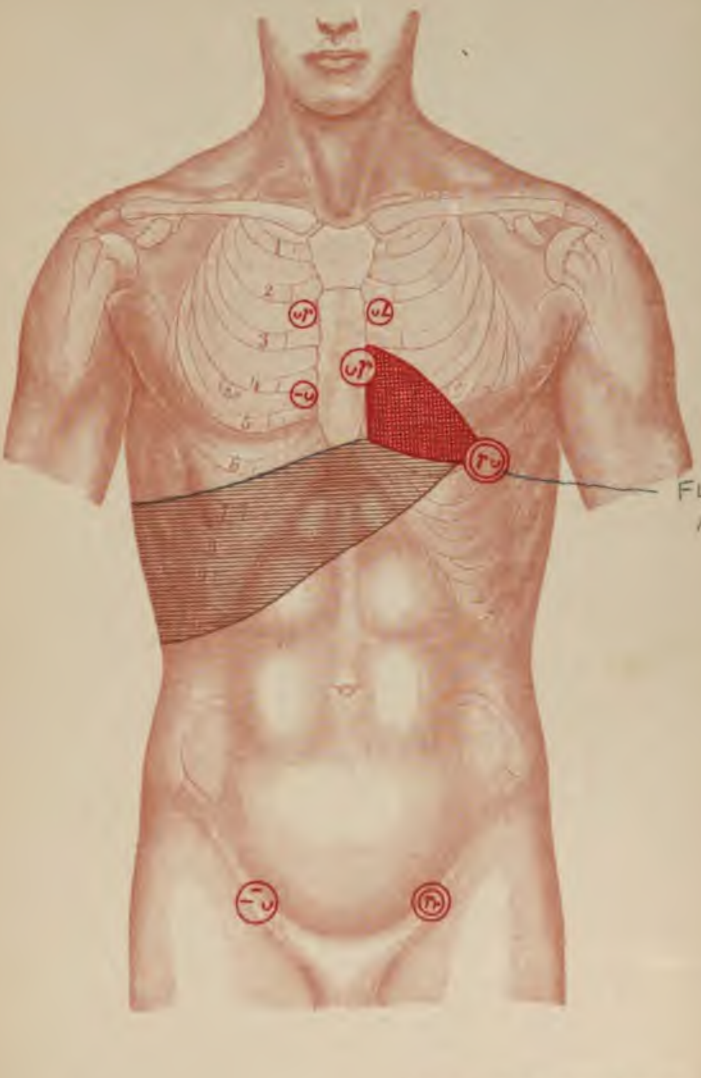
On **Percussion** the upper limit of cardiac dulness was found at the lower border of the third rib; the right limit at the left margin of the sternum; the left limit, two-fingers' breadth external to the left mammillary line. The apex-beat was situated in the fifth and sixth intercostal spaces, two-fingers' breadth external to the mammillary line. It was strongly heaving and extended, on both inspection and palpation.

On **Auscultation** over the aorta the first sound was clear, with a loud rushing diastolic murmur (duk **duch** duk **duch**) (audible in the typical situation, and better still at the left margin of the sternum in the third intercostal space); over the pulmonary artery, duk **duk** duk **duk**; over the mitral orifice (a soft systolic murmur), **duch** duk **duch** duk; over the tricuspid, **duk** duk **duk** duk.

The pulse was running (celer), large, and extremely tense. The radial artery was musical. Double sounds were heard over the femoral artery, and double murmurs upon strong pressure. Capillary pulsation was distinct at the finger-nails.

Diagnosis.—Insufficiency of the aortic valves.

Tab. 46.



FLINT
MURM

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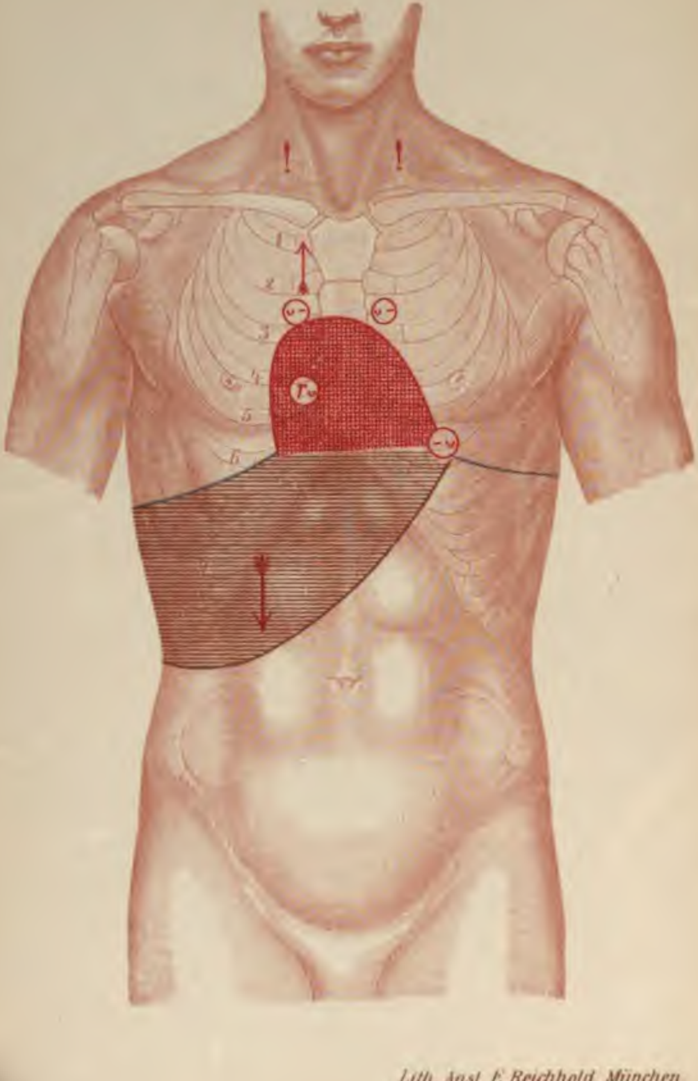

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PLATE 47.

TRICUSPID INSUFFICIENCY.

This cardiac lesion occurs usually in conjunction with other valvular lesions (mitral stenosis, etc.) when compensation becomes deranged. It is here for didactic reasons represented as existing alone.

On **Percussion** the upper limit of cardiac dulness was found at the upper border of the third rib; the right limit 3 cm. (1 in.) to the right of the right margin of the sternum, especially in the third intercostal space (dilatation of the auricle); the left limit somewhat within the mammillary line. The apex-beat was in the mammillary line in the fifth intercostal space.

On **Auscultation**, over the tricuspid orifice was heard a loud, blowing systolic murmur, with a faint first sound, and a clear but faint second sound (**duch** duk **duch** duk). When the first sound became entirely lost the murmur sounded **ch** duk **ch** duk. There were, beside, marked epigastric pulsation and a positive systolic venous pulse (jugular vein, hepatic vein).

Diagnosis.—Insufficiency of the tricuspid valves.

PLATE 48.

IDIOPATHIC HYPERTROPHY OF THE HEART.

History.—A beer-brewer, 46 years old, had complained for several months of increasing respiratory difficulty, cough, sleeplessness, fulness in the head, and attacks of vertigo.

On **Examination** there was found a marked degree of obesity, with a deeply reddened face, watery eyes, and tremulous tongue. There was also a slight degree of pulmonary emphysema.

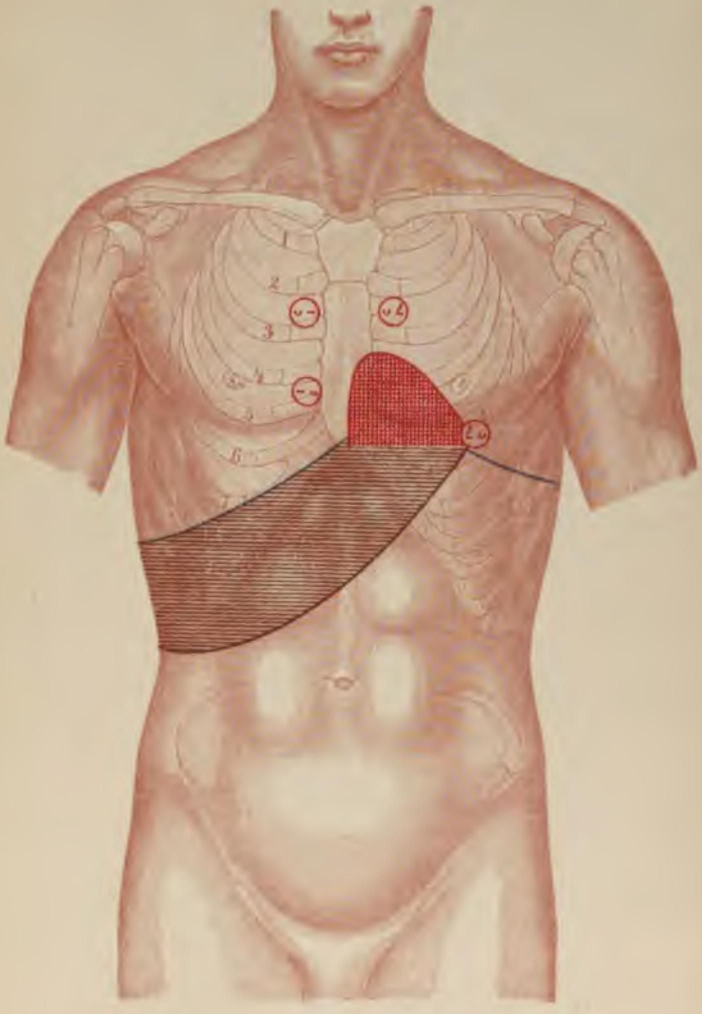
On **Percussion** in the cardiac area, with the patient in the recumbent posture, no distinct result was obtained. The limits of cardiac dulness (upon deep percussion) appeared extended in all directions, and it became more distinct when the patient inclined the body forward.

On **Auscultation** clear but not very loud sounds were heard at all of the cardiac orifices. The pulmonary second sound was accentuated. The apex-beat was in the fifth intercostal space one-finger's breadth external to the mammillary line (most important symptom). Epigastric pulsation was pronounced. The urine was free from albumin.

Diagnosis.—Idiopathic hypertrophy of the heart, with dilatation of both ventricles.

Treatment.—Digitalis; restriction of the amount of fluids (alcoholics).

Tab. 48.



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Tab. 49.

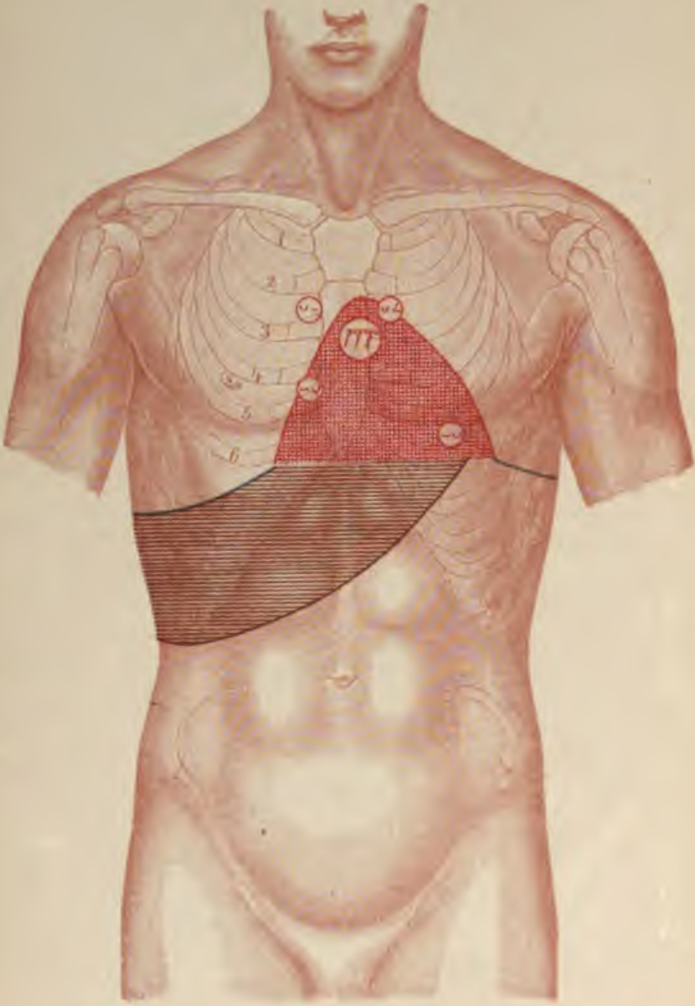

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PLATE 49.

EXUDATIVE PERICARDITIS.

History.—A man, 35 years old, two of whose sisters had died of tuberculosis, had suffered for several weeks from chest-pains and respiratory difficulty. There were some cough and occasional asthmatic attacks.

On **Examination** there were found marked cyanosis and dyspnea, with dry cough. The temperature was 38.7° C. (101.7° F.), the pulse small, frequent (126), and irregular. The area of cardiac percussion-dulness was enormously increased. The upper limit corresponded with the lower margin of the second rib; the right extended beyond the right parasternal line; the left 2 cm. (¾ in.) external to the left mammillary line. The area of percussion-dulness was triangular, with the base below and the truncated apex above.

On **Auscultation** the heart-sounds were only faintly audible, but quite clear. Eight days previously, in association with a smaller area of cardiac percussion-dulness, there had been heard over the upper portion of the sternum a loud rasping quadruple murmur obscuring the heart-sounds—pericarditic friction—which had disappeared. The apex-beat was not strong, but was palpable in the normal situation (that is, within the limits of dulness). Crepitant râles were heard upon the posterior aspect of both lungs posteriorly (compression).

Diagnosis.—Exudative (tuberculous ?) pericarditis.

Treatment.—Powdered digitalis-leaves (0.15—gr. ijss) every two hours; injection of morphin (0.01—gr. ⅙); ice-bag.

PLATE 50.

ANEURISM OF THE AORTA.

History.—A woman, 34 years old, had suffered for five years from palpitation of the heart and pains in the chest upon stooping. Recently there had occurred frequently alarming attacks of shortness of breath. There was no cough.

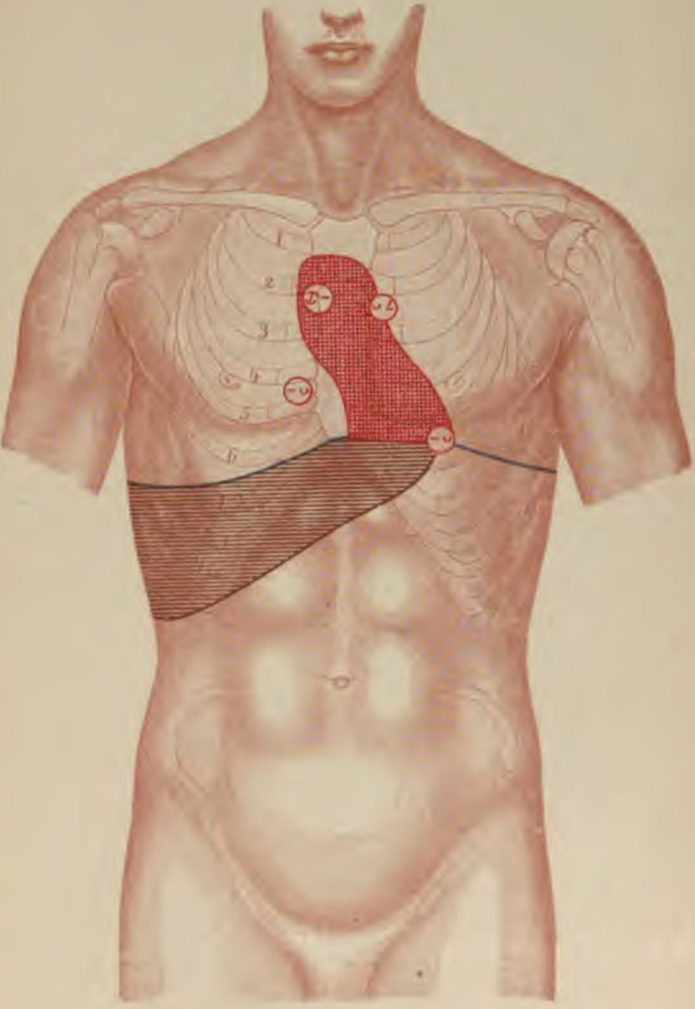
On **Examination** the breathing was found to be noisy and stertorous. The chest-wall over the upper portion of the sternum and the right second intercostal space was distinctly prominent, and to the hand applied in this situation an active pulsatile vibration was palpable.

On **Percussion** the note over the projection was dull in an area as large as the palm of the hand, the dulness merging below with the normal area of cardiac dulness.

On **Auscultation** a soft systolic murmur was heard at the apex of the heart; and at the base a vibratile systolic murmur. The second sound was clear. No difference could be detected between the radial pulses.

Diagnosis.—Aneurism of the ascending portion and of the arch of the aorta.

Tab. 50.



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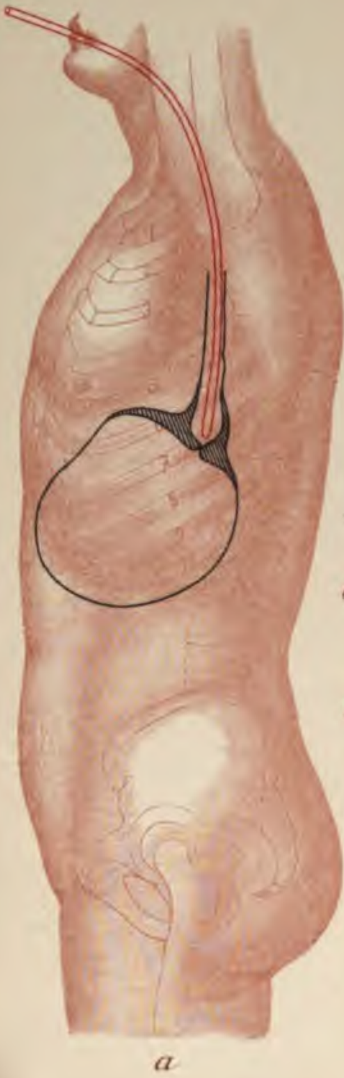
DIAGRAMMATIC REPRESENTATION

OF

DISEASES OF THE ABDOMINAL ORGANS.

(PLATES 51-68.)

Tab. 51.



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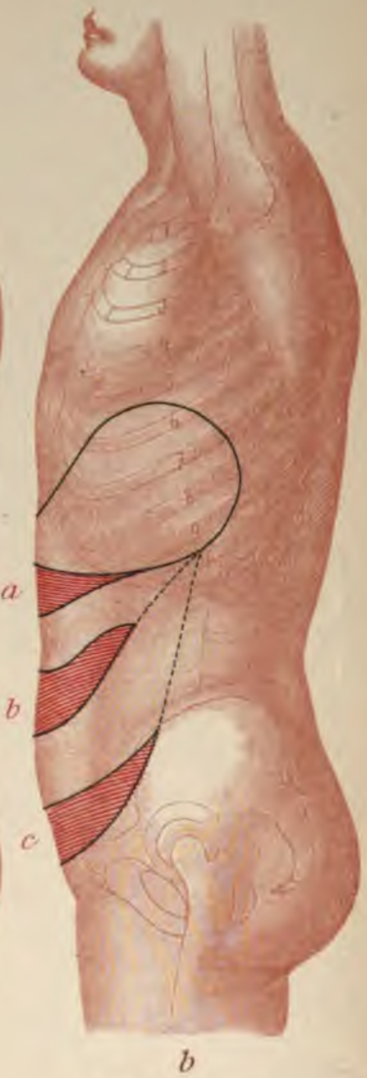

b

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PLATE 51.

Fig. *a.* CARCINOMA OF THE CARDIA, WITH STENOSIS OF THE ESOPHAGUS.

History.—A man, 52 years old, had complained for three months of loss of appetite, emaciation, and vomiting of certain kinds of food. For the same period there had also been some difficulty in swallowing, especially of the more solid articles of food.

On **Examination** the man was found to be greatly emaciated, cachectic, and sallow. Heart and lungs were normal. There were no symptoms of aneurism of the aorta, such as would interdict the use of an esophageal probe. Nothing striking was found in the region of the stomach.

The probe readily entered the esophagus for a distance of 38.5 cm. (15 in.), but at this point it encountered an impassable obstruction.

Diagnosis.—Carcinoma of the cardia.

Fig. *b.* NORMAL AND PATHOLOGIC POSITION OF THE STOMACH.

If the normal stomach be partially filled with water, an oval area of dulness appears 3 cm. (1 in.) above the umbilicus. If the stomach be dilated, this area of dulness forms at or below the level of the umbilicus. In extreme cases the dulness may extend to the symphysis pubis, in accordance with the degree of dilatation.

PLATE 52.

CICATRICIAL STENOSIS OF THE PYLORUS.

History.—A woman, 42 years old, had suffered for ten years with gastric derangement. As a young girl she had, on one occasion, vomited blood (gastric ulcer). From this time she suffered, at varying intervals, from pain in the stomach, particularly after the ingestion of coarser articles of food, together with acid eructation and the vomiting often of large amounts of acid material from four to six hours after meals, following which relief ensued. A considerable degree of emaciation existed.

On **Examination** heart and lungs were found normal, and palpation of the abdomen disclosed no peculiarity.

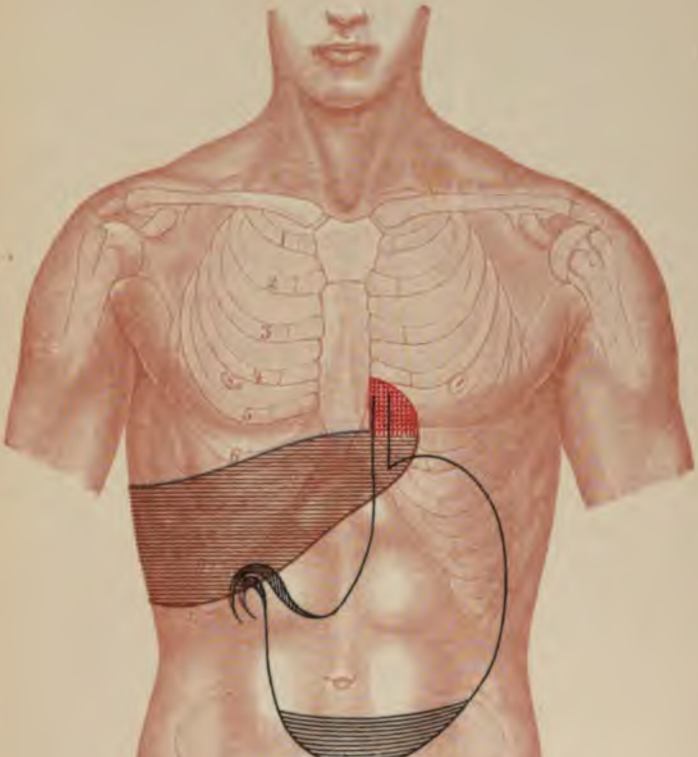
The esophagus permitted the passage of a probe, which entered the stomach for a distance of 60 cm. (23½ in.), its extremity being felt distinctly through the emaciated abdominal walls two-fingers' breath below the umbilicus.

On washing out the fasting stomach in the morning it was found to contain an abundance of fluid, and remains of food. The phloroglucin-vanillin test yielded a positive reaction, the total acidity, estimated as hydrochloric acid, equalling 2.24 per cent. (hyperacidity). A trial-beefsteak had not disappeared from the stomach after eight hours.

Diagnosis.—Cicatricial stenosis of the pylorus; dilatation of the stomach.

Treatment.—Systematic irrigation of the stomach; sodium bicarbonate in small amounts; liquid diet; meat-diet (finely divided meat) carefully cooked. Should the nutrition not improve, operative pyloroplasty.

Tab. 52.



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Tab. 53.

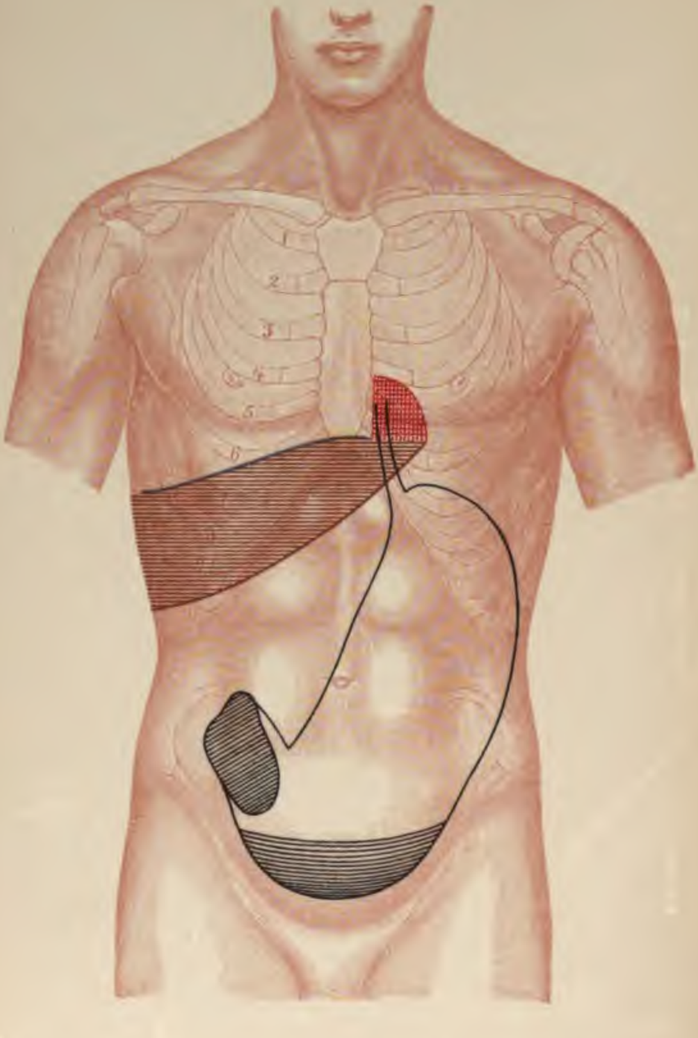

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PLATE 53.

DILATATION OF THE STOMACH, WITH CARCINOMA OF THE PYLORUS.

History.—A man, 52 years old, had suffered for many years from gastric derangement, etc. (See history appended to Plate 52, with which this closely agrees.)

On **Examination** the lower portion of the abdomen below the umbilicus was found markedly prominent, especially upon the left side, the projection having entirely the form of the contour of the stomach. On protracted observation distinct slow, vermicular, creeping waves of contraction could be seen to pass over the prominence from left to right. On percussion loud splashing sounds were generated. Upon the right side below the umbilicus palpation disclosed the presence of a hard nodular tumor as large as an apple, and capable of a considerable degree of mobility. Lavage of the stomach yielded evidence of marked stagnation of the gastric contents, no free hydrochloric acid being demonstrable in the washings after a trial-breakfast, although the reaction for lactic acid was distinctly present. When the stomach was partially filled with water the previously tympanitic note above the symphysis pubis was displaced by a semilunar area of dulness.

Diagnosis.—Dilatation of the stomach following pyloric stenosis (resulting from a cicatrix following ulceration); secondary carcinoma.

Treatment.—Operative removal (extirpation of the pylorus, gastro-enterostomy).

PLATE 54.

CARCINOMA OF THE STOMACH.

History.—A man, 52 years old, previously healthy, had noticed for about three months that he was losing flesh. The appetite became impaired, and there was often acid eructation and occasionally vomiting. He could not stand as much exertion as formerly, and he felt tired and languid.

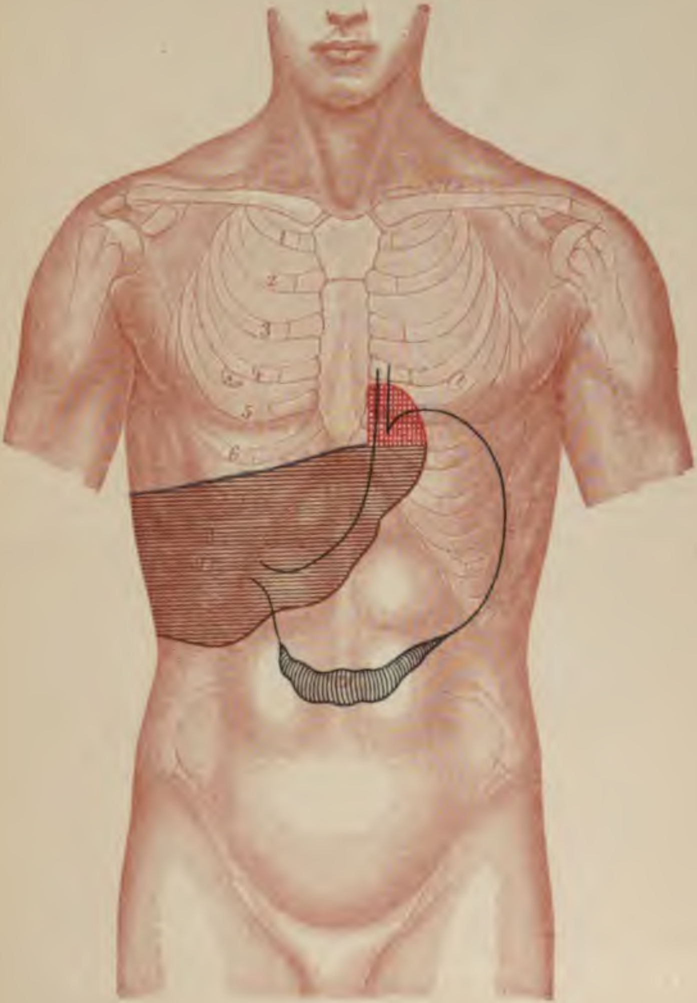
On **Examination** the patient was found to be considerably emaciated, and presenting the carcinomatous cachexia (sallow pallor of the face) and marked anemia. Heart and lungs were normal. In the epigastrium a hard, nodular, sausage-shaped tumor, lying transversely, was felt through the thin abdominal walls at the level of the umbilicus. The mass was somewhat sensitive to pressure, and it moved but little with respiration. In addition similar hard tumors were felt at the costal margin on the right side. These could be followed beneath the costal arch up toward the liver, and descended 2 cm. (¾ in.) with inspiration. The liver-dulness merged with the dulness over these tumors.

On washing out the stomach after a trial-breakfast no free hydrochloric acid was demonstrable in the gastric contents, although there was a pronounced reaction to tests for lactic acid.

Diagnosis.—Carcinoma of the greater curvature of the stomach; metastasis to the liver.

Treatment.—Fluid and semi-fluid diet, eggs, milk; after eating always, several times daily, from five to ten drops of dilute hydrochloric acid in water; extract of condurango.

Tab. 54.



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Tab. 55.

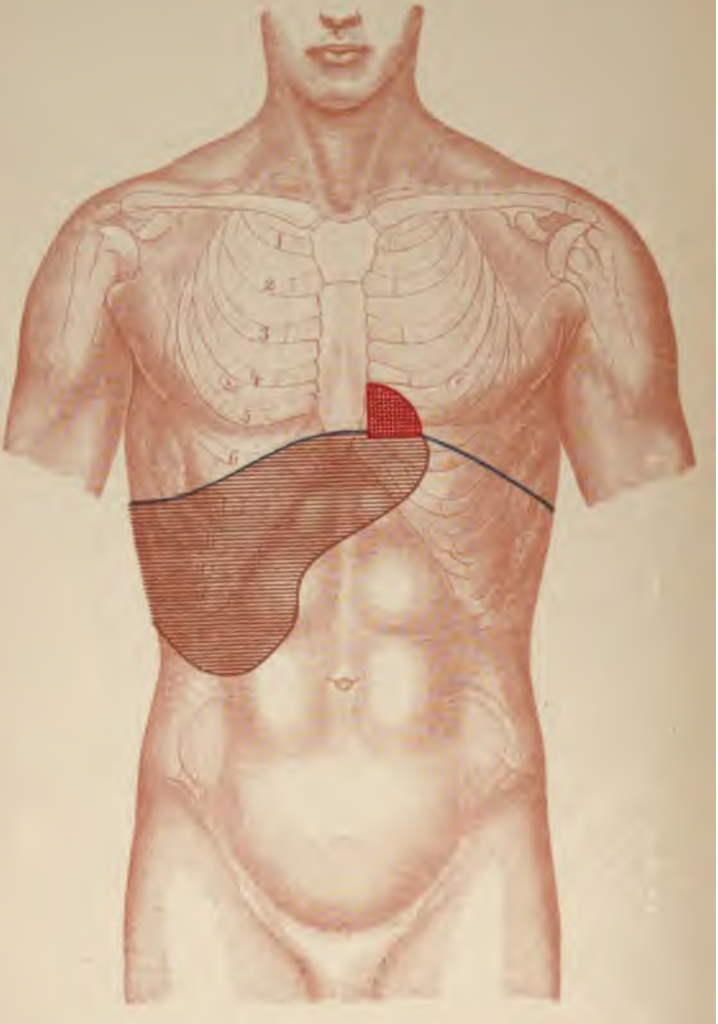

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PLATE 55.

CONSTRICTED LIVER.

History.—A woman, 43 years old, had complained for years of occasional severe pain in the right side and in the region of the stomach. Stooping was especially difficult. At times there was vomiting, and there were other nervous symptoms.

On **Examination**, when the abdominal walls were relaxed, there was felt, with great distinctness, below the costal margin, on the right side, a smooth tumor of firm consistence, disappearing above under the costal arch, and which could be grasped from below with the tips of the palpating fingers, and could be folded over upward.

On **Percussion** the liver-dulness was found to merge with that over the tumor.

Diagnosis.—Constricted right lobe of the liver.

PLATE 56.

CIRRHOSIS OF THE LIVER.

History.—A hotel-keeper, 48 years old, previously always healthy and accustomed to the generous use of alcohol, had lost considerable flesh during several months. The appetite was impaired, the abdomen was greatly distended, the lower extremities were swollen, and there were respiratory difficulty, sleeplessness, and frequent diarrhea.

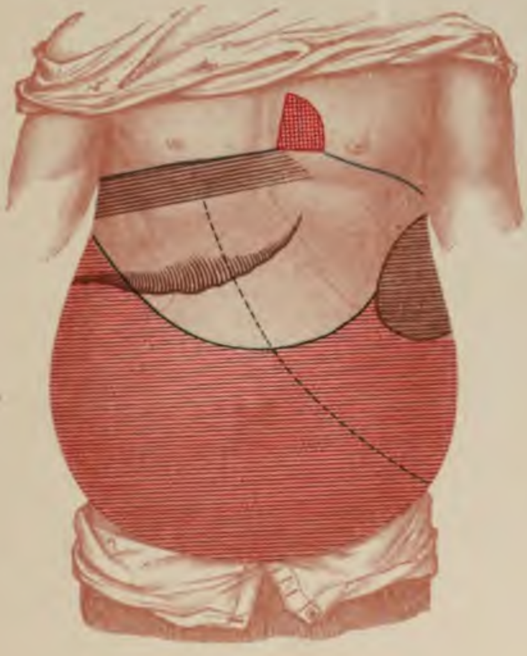
On **Examination** the patient was found considerably emaciated, with a tremulous, coated tongue, edema of the lower extremities, and marked dyspnea. The temperature was normal, the pulse 116 and small and not quite regular. The diaphragm was displaced upward (the lower limit of the right lung was found at the upper border of the fifth rib and the area of cardiac percussion-dulness was higher than normal). The abdomen was greatly distended and enlarged laterally. Palpation disclosed tensely distended abdominal walls; and upon percussion fluctuation was distinctly demonstrable.

On **Percussion** a tympanitic note was found only in the middle of the abdomen above the umbilicus. All other dependent portions were absolutely dull, the upper limit of dulness (black) pursuing a semicircular course. The area of liver-dulness was very small, scarcely demonstrable. Upon the left the large area of dulness merged with that of the spleen. When the patient lay upon his right side the limit of dulness changed (as shown by the interrupted black line), while the percussion-note on the left side became tympanitic and the area of splenic dulness was found to be distinctly enlarged.

Diagnosis.—Free effusion into the peritoneal cavity (ascites); probably cirrhosis of the liver.

Treatment.—Puncture, with the evacuation of 12 liters (quarts) of clear, light-yellow fluid (containing little albumin). The lower margin of the liver could now be felt to be distinctly hard, nodular, and contracted.

Tab 56.



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Tab. 57.

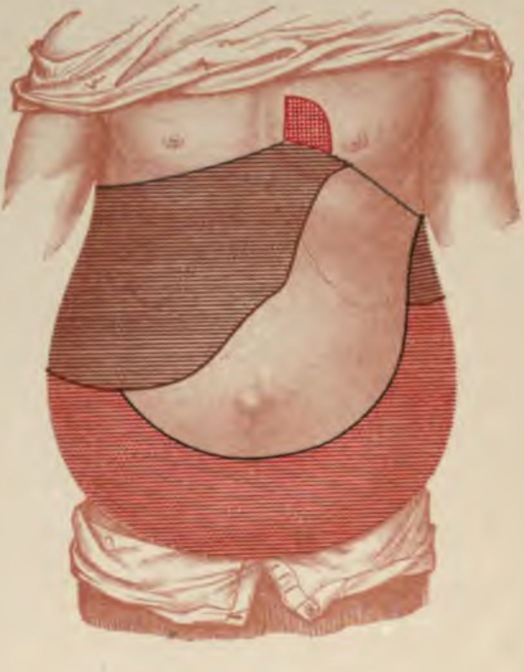

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PLATE 57.

MELANOSARCOMA OF THE LIVER.

History.—A man, 50 years old, had had his left eyeball enucleated eight weeks previously on account of a malignant growth (melanosarcoma of the choroid). For four weeks he had noticed that his abdomen was swollen and tense and he had grown thinner and weaker.

On **Examination** there were found distinct icteric discoloration of the skin and a marked degree of emaciation. The lungs and heart exhibited no noteworthy alteration. The diaphragm was displaced upward.

The abdomen was exceedingly prominent, its walls greatly distended. In its right half, projecting beneath the costal margin, was felt a large, solid, hard tumor, with a nodular surface. The mass reached downward to the level of the umbilicus and presented a round margin. It occupied the entire epigastrium down to the umbilicus. Palpation was attended with considerable pain.

The area of hepatic percussion-dulness began as high as the upper border of the fifth rib and merged with that of the tumor. In the dependent portions of the abdomen there was everywhere dulness (red), which varied with change in posture (ascites). The area of splenic percussion-dulness was not increased.

The urine yielded positive reactions to tests for biliary pigment and for melanin. (See Fig. 11, Plate 20.)

Diagnosis.—Metastatic melanosarcoma of the liver; ascites.

Treatment.—Infusion of digitalis (1½ to 120—gr. xxij to 4 oz.) and morphin.

Upon post-mortem examination three weeks later the liver was found to be enormous in size (42 cm. long—16 in.) and the seat of innumerable sarcoma-nodules.

PLATE 58.

CHOLELITHIASIS; HYDROPS OF THE GALL-BLADDER.

History.—A woman, 44 years old, had suffered for four years from periodically recurring attacks of colic in the region of the liver and the stomach, with severe vomiting and at times jaundice. Of late the attacks of colic were not rarely attended with severe chills. On the night before coming under observation there had occurred an attack of colic of unusual severity, lasting several hours, and requiring morphin subcutaneously.

On **Examination** the patient was found to be exhausted and somewhat emaciated. Jaundice was distinct, the sclera being yellow and the urine dark brown in color. The temperature was 38.5° C. (101.3° F.), the pulse 104.

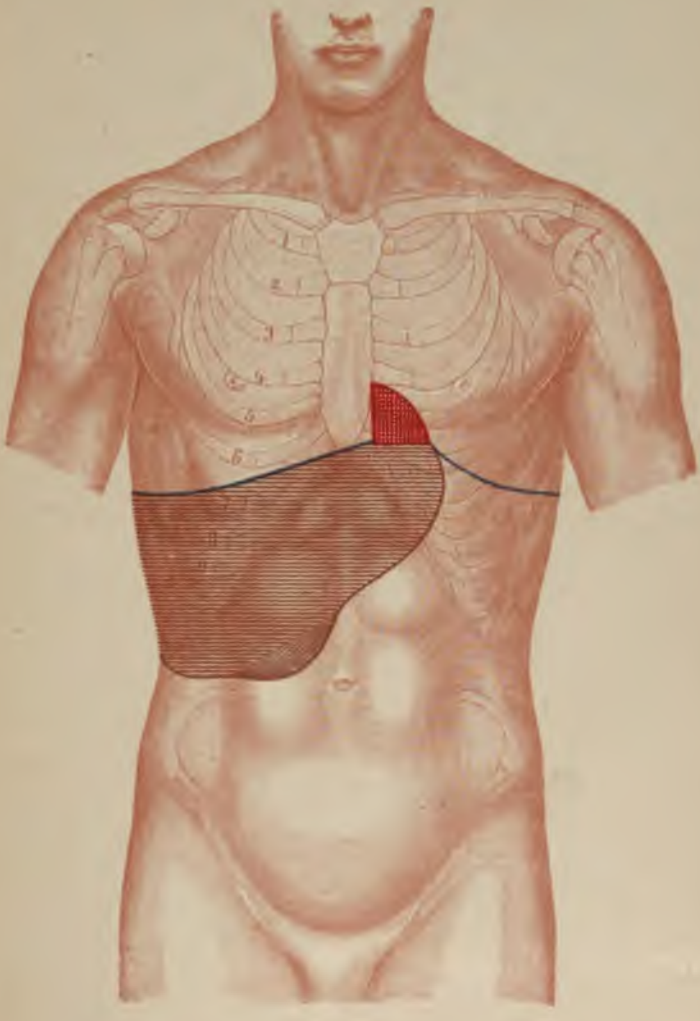
On **Palpation** the abdomen was found to be painful, and pressure in the region of the liver induced complaint of suffering. Beneath the right half of the costal arch in the mammillary line was readily felt a hard, tensely distended tumor, with a smooth surface, of the size of a billiard-ball, extending almost to the umbilicus.

The lower margin of the liver was distinctly palpable throughout its whole extent, crossing the tumor described and extending three-fingers' breadth beyond the costal arch.

Diagnosis.—Cholelithiasis; recent impaction of a calculus in the choledoch duct; ancient occlusion and cicatrix-formation in the cystic duct; hydrops of the gall-bladder.

Treatment.—Opium, poultices, later cholecystotomy.

Tab. 58.



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Tab. 59.

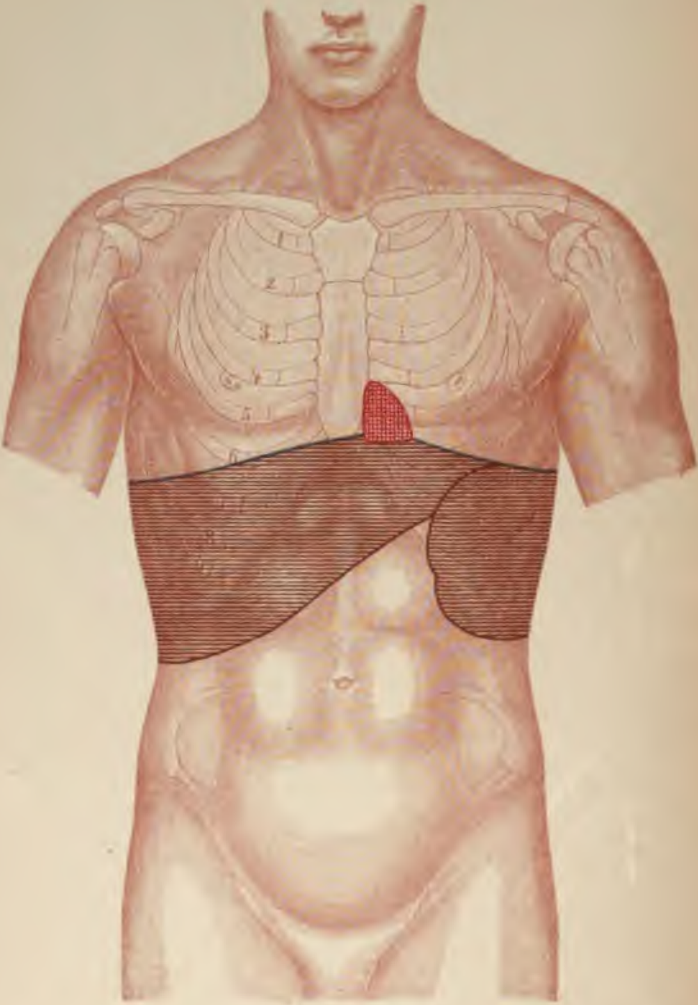

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PLATE 59.

AMYLOID DEGENERATION.

History.—A girl, 18 years old, with congenital syphilis, had had for eight years an extensive perforation of the nasal septum (saddle-nose). For ten months there had been a free discharge of pus and crusts from the nose, with empyema of the maxillary antrum and purulent catarrh of the middle ear (for a long time untreated). For eight days there had been marked swelling of the lower extremities, and for a considerable time diarrhea.

On **Examination** the patient was found to be extremely pale and greatly emaciated. The face was puffy, and the nose presented the condition described. There were, besides, edema and dyspnea.

The lungs exhibited bronchitic râles over the lower lobes. The heart displayed no abnormality.

The abdomen was not distended and there was no ascites.

The liver extended two fingers' breadth beyond the costal arch, and its free margin was rounded. Its consistence was uniformly firm and its surface was smooth.

The spleen was distinctly palpable and of hard, dense consistence. The dulness extended 1 cm. (⅓ in.) beyond the costal margin.

The urine was fairly abundant and pale, and contained but little sediment and much (½ volume per cent.) albumin.

Diagnosis.—Amyloid degeneration of liver, spleen, kidneys, and intestines.

Treatment.—Diuretics; surgical treatment of the suppurative processes.

PLATE 60.

SPLENO-MEDULLARY LEUKEMIA.

History.—A man, 35 years old, previously always healthy had for six months not felt so well as formerly. His appetit was impaired, and he readily became tired and short of breat on walking. There was almost constant pain in the left side and the abdomen had become larger than before, while othe parts of the body had wasted.

On **Examination** the patient was found to be pale an emaciated. The temperature was 38.2° C. (100.8° F.) (in th evening), the pulse 92.

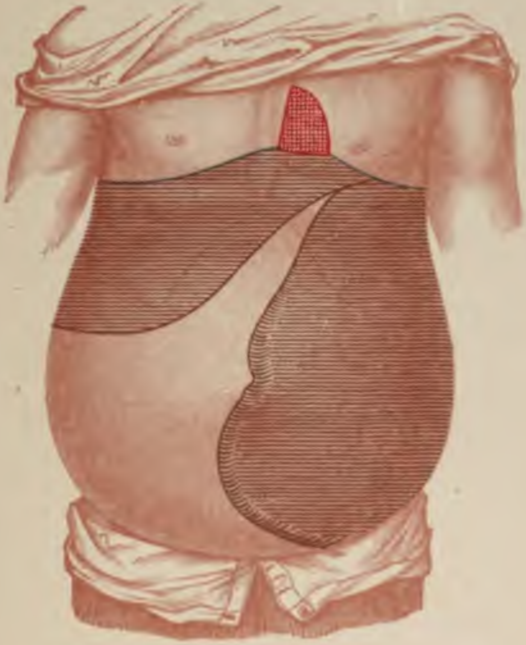
The lungs presented no abnormality. A systolic murmur wa audible over the apex-beat of the heart. The abdomen wa greatly distended, the left side more so than the right, and th abdominal walls were exceedingly tense. In the left half of th abdomen was found a firm, hard tumor, extending almost t the symphysis pubis, being lost to the left beneath the costa arch, and extending to the right somewhat beyond the middl line. At the level of the umbilicus a distinct indentation wa palpable in the border of the tumor (incisura), which was otherwise rounded and smooth. The dulness over the tumor merge with that of the spleen. The area of hepatic percussion-dulness was likewise increased, extending four-fingers' breadt beyond the costal margin. The liver could be felt to be hard but with a sharp margin. In the dependent portions of th abdomen upon the right the percussion-note was tympaniti and clear (no ascites).

Blood-examination disclosed a leukemic state. (See Plate 4 Figs. 1 and 2.)

Diagnosis.—Spleno-medullary leukemia (after blood-examination).

Treatment.—Arsenic.

Tab. 60.



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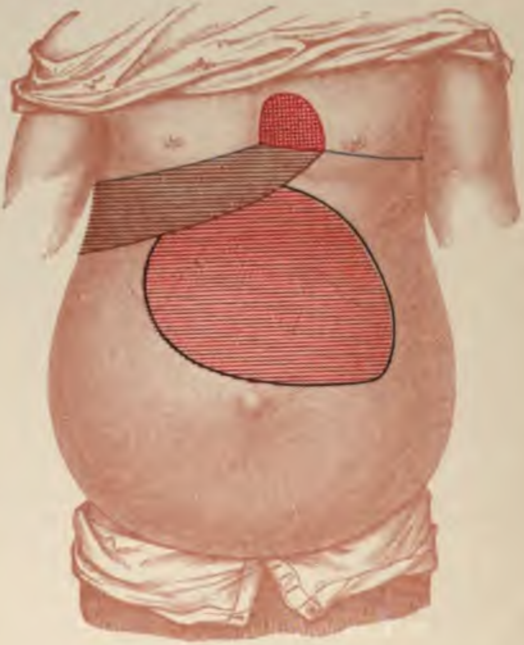

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PLATE 61.

PANCREATIC CYST.

History.—A girl, 5 years old, always weakly and pallid, had for several weeks been taking less nourishment than previously and vomited frequently. During this time the abdomen had become greatly swollen, and for several days the child was unable to stand. Breathing was difficult and accelerated.

On **Examination** the child was found to be anemic and emaciated. Weakness was pronounced. The temperature was normal, the pulse 140. Dyspnea was marked.

The abdomen was immensely distended and the abdominal walls exceedingly tense.

The epigastrium was especially prominent, and was occupied by a tense, elastic tumor with a smooth surface and as large as a child's head. The dulness over the mass merged above and to the right with that of the liver, while below and to the left it was readily separable from the tympanitic note of the bowel.

Splenic and hepatic percussion-dulness was not increased. The diaphragm was displaced upward and the area of cardiac percussion-dulness increased (dilatation). Exploratory puncture of the tumor evacuated clear, yellowish fluid containing little albumin and sodium chlorid and free from morphologic elements (no echinococcus-hooklets).

Diagnosis.—Cystically degenerated pancreas.

Treatment.—Operative exploration (which was declined by the relatives).

Postmortem examination (twelve days later) confirmed the diagnosis.

PLATE 62.

ASCITES ATTENDING CARDIAC LESION.

History.—A woman, 48 years old, had suffered for a number of years from a valvular lesion of the heart and had repeatedly experienced derangements of compensation. For two weeks the shortness of breath had again increased considerably, and sleep and appetite had become poor. The lower extremities had been for some time considerably swollen and the abdomen was gradually increasing in size. The patient could get air enough only with difficulty, by sitting at the edge of the bed, resting upon the arms.

On **Examination** there were seen to be marked cyanosis and dyspnea (orthopnea), with edema of the lower extremities.

The pulse was scarcely palpable and extremely irregular. The lungs were compressed by effusion into the pleural cavities and the heart was greatly dilated, etc. The diaphragm was displaced upward.

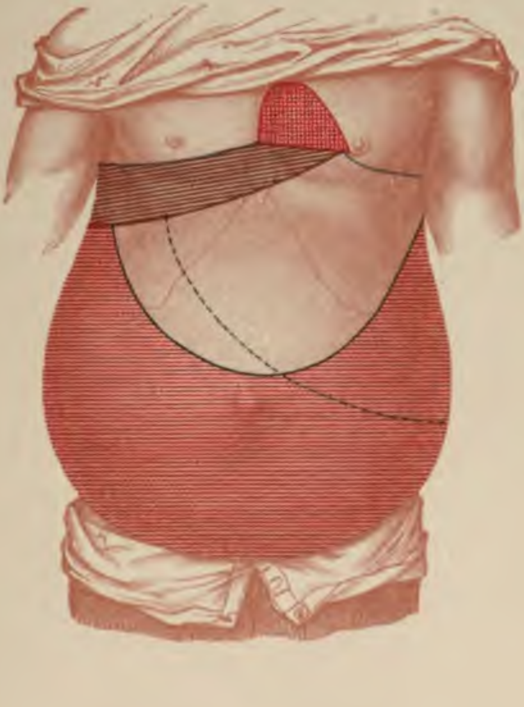
The abdomen was found to be greatly and uniformly distended and enlarged. The area of hepatic percussion-dulness was diminished, although the liver could on palpation be felt to be enlarged.

In the dependent portions of the abdomen the percussion-note was absolutely dull, with a sense of resistance. There was well-marked fluctuation. The upper concave limit of dulness (black line) occupied the middle line at the level of the umbilicus, while above the note was tympanitic. When the patient lay upon her side the limits of dulness changed (see interrupted black line); lying upon the right side, for instance, the dulness on the left was replaced by a tympanitic note, while the area of splenic dulness appeared distinctly enlarged, reaching to the costal margin, where the spleen could be felt. The urine was concentrated and reacted to tests for albumin.

Diagnosis.—Ascites; cyanotic liver, spleen, and kidneys; ruptured compensation of a cardiac valvular lesion.

Treatment.—Powdered digitalis 0.15 (gr. ijss), calomel 0.2 (gr. iij), every three hours.

Tab 62.



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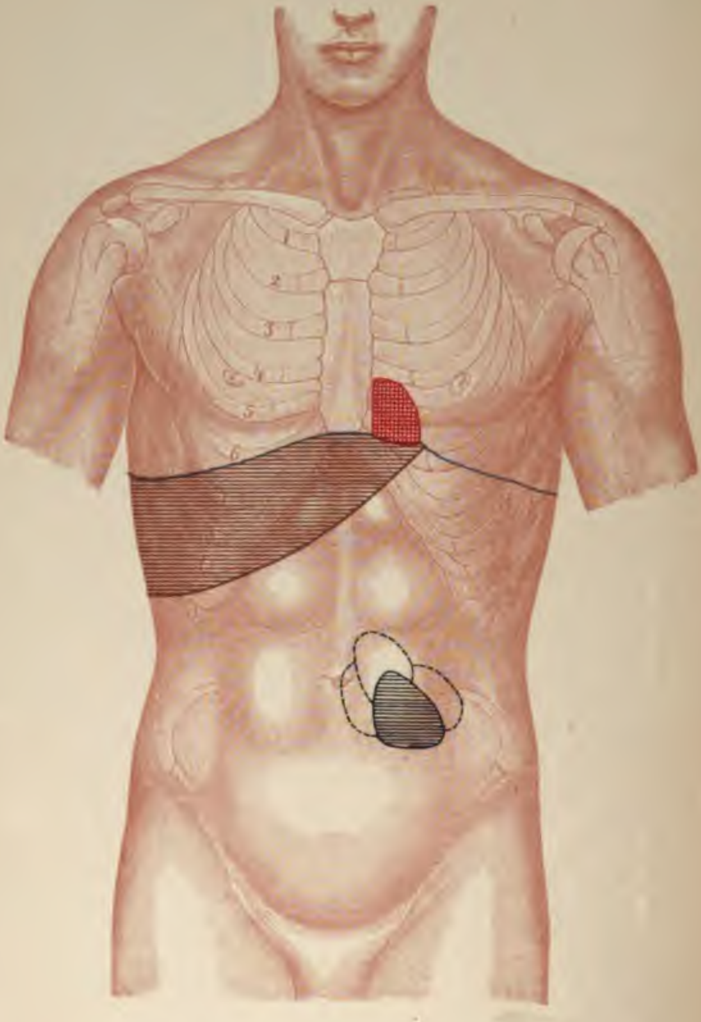

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PLATE 63.

CARCINOMA OF THE INTESTINE.

History.—A man, 50 years old, had been conscious for several months of dull, heavy pain in the abdomen. The appetite was slight, the bowels constipated, and vomiting was frequent.

On **Examination** heart and lungs were found normal.

In the left half of the abdomen, somewhat below the umbilicus, a firm nodular tumor as large as an apple could be felt. This mass was immovable with respiration, but with the hand could be displaced a considerable distance to right, to left, and upward (in slighter degree downward).

There was no swelling of the liver, and the spleen was not palpable. The lymphatic glands were not enlarged. In the gastric juice, after a trial-breakfast, hydrochloric acid was readily demonstrable (therefore no carcinoma of the stomach).

Diagnosis.—Carcinoma of the small intestine.

Treatment.—Operative removal (which was successfully effected and the diagnosis confirmed).

PLATE 64.

OCCLUSION OF THE BOWEL; INVAGINATION.

History.—A man, 42 years old, was seized with abdominal pain, eructation, and nausea, and for two days had suffered from constipation. Soon severe and repeated vomiting set in and the abdominal pain became intense.

On **Examination** the patient was found to be a robust man, who lay in bed doubled up and groaning with pain. From time to time there were severe hiccough and nausea, with vomiting of bile-stained mucus of feculent odor. No hernia could be detected.

The abdomen was considerably distended and tender upon manipulation (palpation was preceded by an injection of morphin). Through the abdominal walls a number of distended loops of bowel could be distinctly detected, exhibiting peristaltic movement. On the right side at the level of the umbilicus there could be felt a sausage-shaped tumor, tensely distended and readily movable in all directions. In the dependent portions of the abdomen the percussion-note was impaired.

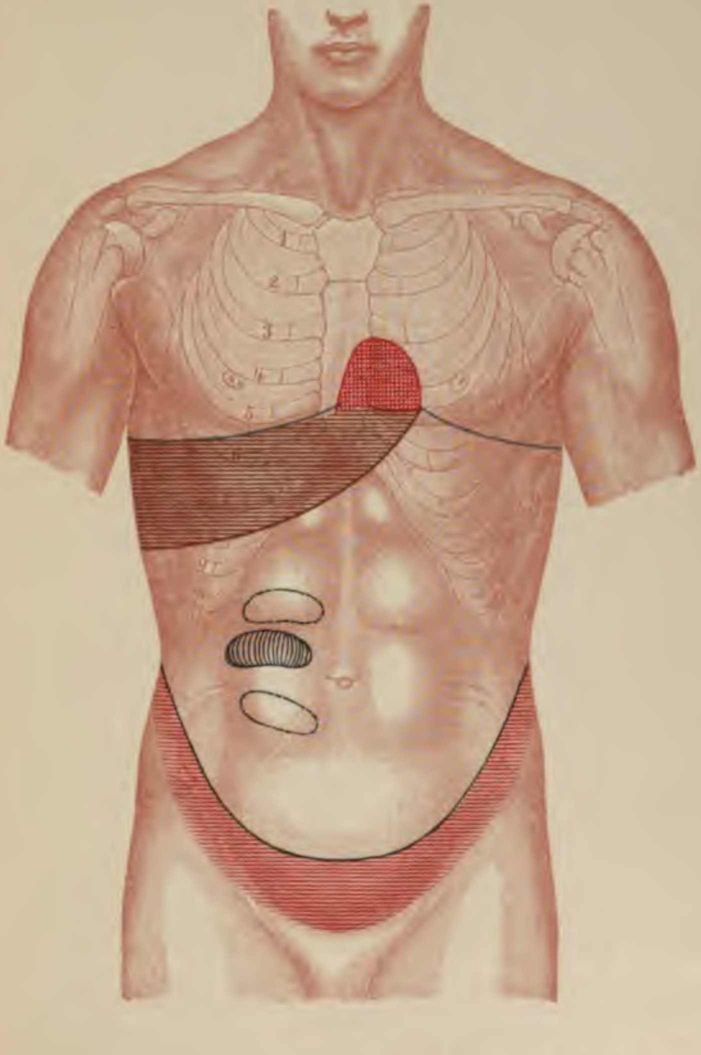
The area of hepatic percussion-dulness was diminished (meteorism). The diaphragm was elevated.

An enema led to the evacuation of fecal matter mixed with bloody, mucous masses.

Diagnosis.—Intestinal occlusion; invagination of the small intestine.

Treatment.—Opium, enemata, lavage of the stomach, celiotomy.

Tab. 64.



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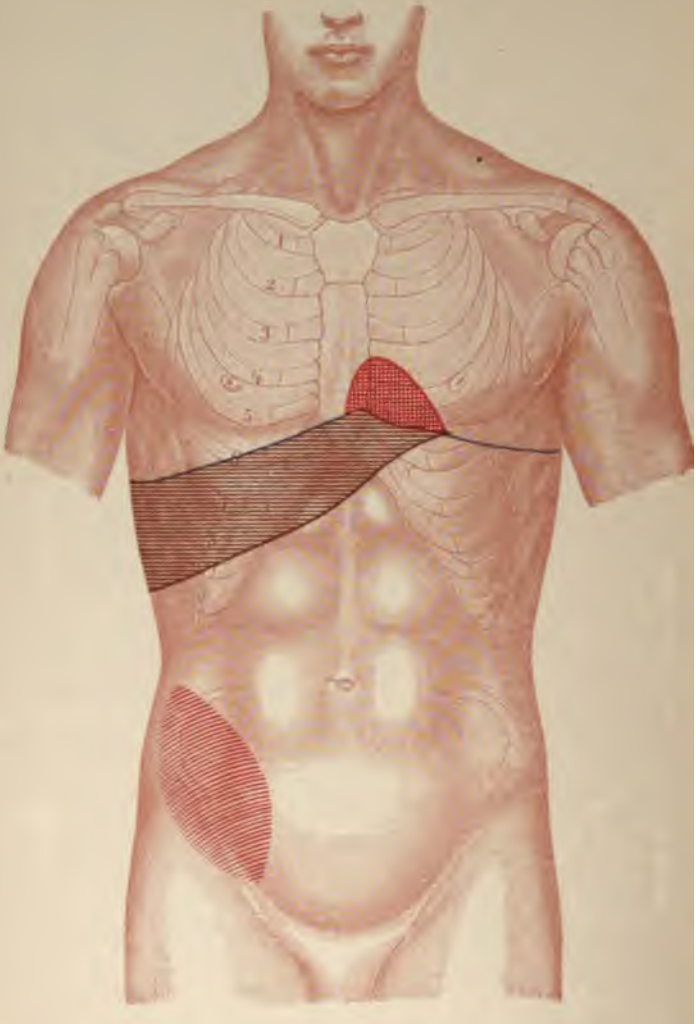

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PLATE 65.

PERITYPHLITIS.

History.—A man, 21 years old, was suddenly seized with severe pain in the abdomen, together with vomiting. The bowels had been perfectly regular. Every movement was attended with intense pain, especially upon the right side. During the night there occurred repeated vomiting.

On **Examination** the temperature was found to be 38.8° C. (101.8° F.), the pulse 104, and strong. The abdomen was greatly distended; the patient twitched upon slightest touch. On palpation a firmly resistant area, about a hand's breadth in extent, was found in the right iliac fossa, limited to the left and above by a sharp border, over which (on gentle percussion) the note, was found to be dull. Vomiting recurred frequently. Other organs presented no abnormality. The urine contained a small quantity of albumin and yielded the reaction for indican.

Diagnosis.—Perityphlitis from perforation of the vermiform appendix.

Treatment.—Ice-bag, tincture of opium (twenty drops every two hours), small amounts of milk and broth.

Course.—During the following days there was slight fever and the pulse was good. On the fourth day there was an exacerbation of the fever, with vomiting. The resistant area had enlarged in an upward direction. Exploratory puncture disclosed the presence of disorganized pus.

Treatment.—Operative evacuation of the abscess, resection of the perforated vermiform appendix.

PLATE 66.

TUBERCULOUS PERITONITIS.

History.—A man, 66 years old, had suffered for eight weeks from loss of appetite, with at times frequent vomiting and diarrhea, as well as persistent abdominal pain. During this time he had emaciated greatly, and for two weeks had been compelled to remain abed. There had for a long time been some cough.

Upon **Examination** there was found a high degree of emaciation, with marked edema of the lower extremities. The temperature was 36.8° C. (98.2° F.), the pulse 126, very small and irregular. Over the apex of the right lung the percussion-note was distinctly impaired, with enfeebled breathing and coarse râles. The diaphragm was elevated.

The abdomen was greatly distended (having a circumference of 104 cm. (41 in.) at the level of the umbilicus). The percussion-note was tympanitic only in the epigastrium; elsewhere it was dull. A distinct sense of fluctuation could be detected. The sputum contained tubercle-bacilli.

Exploratory puncture disclosed the presence of a hemorrhagic exudate.

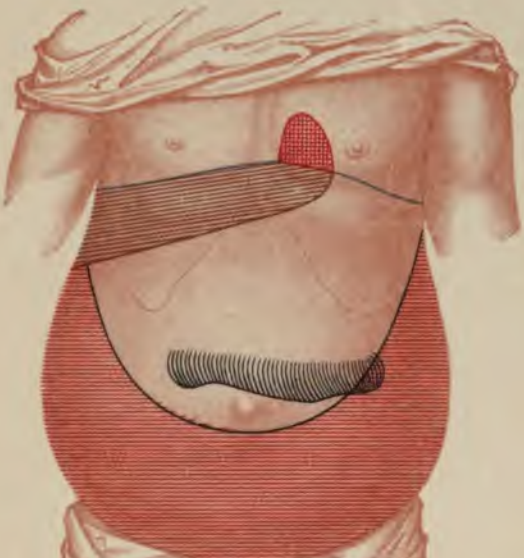
Diagnosis.—Tuberculous peritonitis.

Treatment.—Celiotomy was waived on account of the debilitated state of the patient.

On tapping eight quarts of deeply blood-stained fluid were evacuated.

Following this procedure thick, nodular tumor-masses of a hand's breadth extent were palpable at the level of the umbilicus, and were somewhat movable (tuberculosis of the peritoneum).

Tab. 66.



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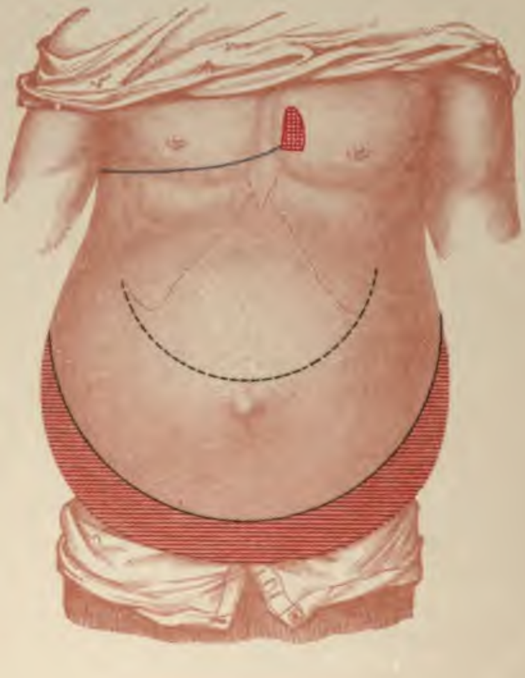

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PLATE 67.

PERFORATIVE PERITONITIS.

History.—A man, 27 years old, had suffered for a number of years from symptoms of chronic gastric ulceration (vomiting, on one occasion hemoptysis, acid eructations, gastric pain). After a mid-day meal he was suddenly seized with severe abdominal pain, with loss of consciousness and in the course of the following hours persistent vomiting and pain.

On **Examination** on the following day (immediate celiotomy having been declined) the patient was found to be in a state of collapse, the nose pointed, the face pale, and perspiration upon the brow. There occurred repeated bilious vomiting, with hiccough. The temperature was 36.5° C. (97.7° F.), the pulse very small—146.

The abdomen was greatly distended, like a balloon, and all manipulation (in spite of the administration of opium) caused exquisite pain. In the dependent portions of the body there was a hand's breadth area of dulness on percussion. The hepatic percussion-dulness was lost and replaced by a loud, tympanitic note. On agitation a distinct metallic splashing sound was elicited. When the patient lay upon the left side the splenic dulness was replaced by a loud, tympanitic note (accumulation of air).

Diagnosis.—Perforation of a gastric ulcer, with rupture into the peritoneal cavity, entrance of air, and purulent peritonitis.

Treatment.—Celiotomy now entirely hopeless; morphin, camphor. Death after three hours.

PLATE 68.

SARCOMA OF THE RIGHT KIDNEY.

History.—A child, 4 years old, had been losing flesh rapidly for two months, at first without appreciable cause. It was restless at night and complained of abdominal pain. The abdomen was always greatly distended. On one occasion the urine was found to be deeply bloody.

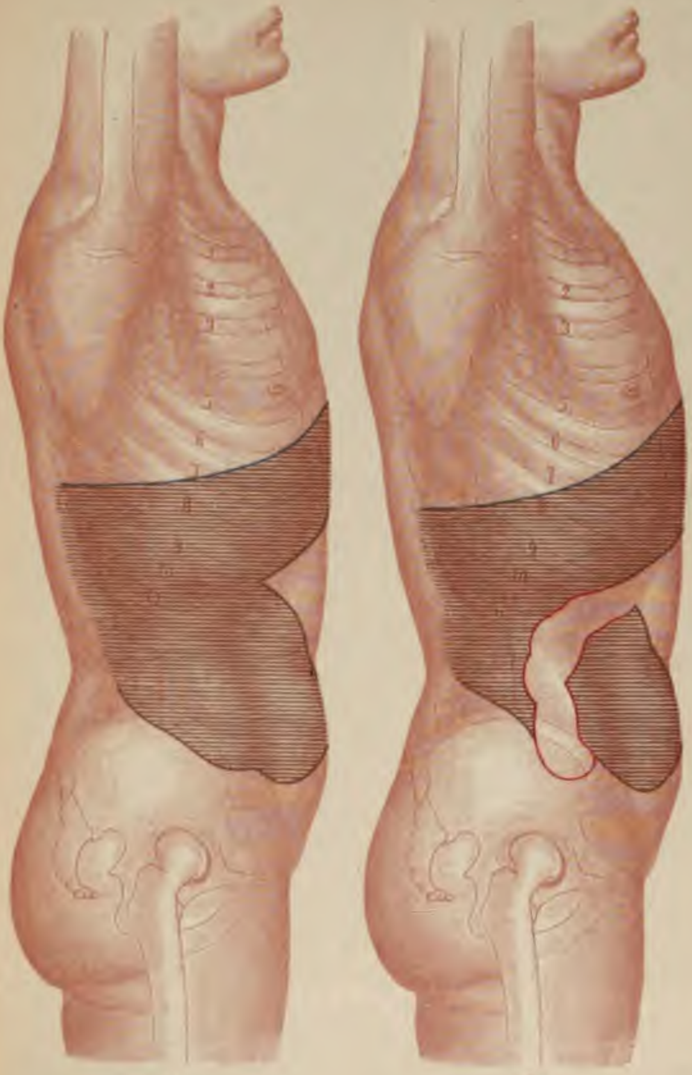
On **Examination** the child was found to be greatly emaciated and anemic. The temperature was normal, the pulse 144. The lungs exhibited no abnormality. The abdomen was protuberant, particularly upon the right side. In this situation could be felt a firm, smooth tumor extending from the lumbar region beneath the costal arch and forward to the umbilicus. This mass was not movable with respiration. The area of hepatic percussion-dulness merged laterally with that of the tumor, not exceeding its normal limits toward the middle line, where the liver could not be felt to be enlarged.

After administration of castor-oil and resulting copious evacuation of the bowels there appeared (Fig. b) from above downward within the previous area of absolute dulness a small band-like zone of tympanitic percussion (ascending colon). There was hematuria, and vermicular blood-clots were expelled with the urine.

Diagnosis.—Sarcoma of the right kidney.

Treatment.—Attempt at extirpation.

Tab. 68.



a b

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SECTION I.

EXAMINATION OF THE PATIENT.

In order to be of service to a patient it is essential to recognize the ailment from which he suffers, and to this end systematic clinical investigation is necessary. This is divided into two parts:

1. The *history*, the *previous state*, as elicited by inquiry.
2. The *present state*, as determined by systematic examination.

Upon both of these is based the *diagnosis*.

The **history** includes:

a. The antecedents and the previous life of the patient from the medical point of view;

b. The origin and the development of the existing morbid state.

The history is obtained by carefully directed inquiry on the part of the physician.

Before taking up the history in detail it is well to attempt to gain by a few questions an approximate idea as to the probable seat of the disease.

Important conclusions as to the nature of the existing disorder may sometimes be reached from a knowledge of certain historic data, and these are herewith mentioned:

1. **Personal.**—*a*. **Age.**—Some diseases occur with preference at certain periods of life:

Diseases of infancy (diarrhea, intestinal catarrh, disorders of dentition, rachitis, eclampsia);

Diseases of childhood (acute exanthemata, measles, scarlet fever, chicken-pox, Rötheln, whooping-cough, diphtheria, scrofulosis, eczema, certain nervous diseases);

Diseases of puberty (chlorosis, tuberculosis, nervous disorders);

) Syph. Chancre where, Tr., how long. Hist. of Rash & throat, & inunctions (Hard or soft 3 wks or more, 3 ds to 3 [illegible] incubation) Gon. No. of times. How long after coitus. Tr., cured (6 wks.) Injections. Complic. (Epid)

Diseases of adult life (venereal diseases, pneumonia, metabolic disturbances, gout, diabetes, obesity; alcoholism); Rheumatism, Malaria, Typhoid.

Diseases of advanced life (carcinoma, arteriosclerosis, deforming arthritis, emphysema, etc.).

b. **Sex.**—The disposition to many diseases as related to sex is variable:

Males (emphysema, hypertrophy of the heart, alcoholism, injuries and their sequelæ, occupation-diseases, etc.).

Females (menstrual disorders, puerperal disorders and their sequelæ, chlorosis, constipation, hysteria).

c. **Occupation.**—See below.

2. **Heredity.**—Hereditary predisposition is especially of significance with relation to the following diseases: tuberculosis (of lungs, glands, bones, joints), syphilis, nervous diseases (neurasthenia, hereditary system-degenerations, mental disorders), diabetes, gout, neoplasms (carcinoma, sarcoma), hemophilia, etc. Alcohol, Rheumatism

3. **Social Relations.**—Careful inquiry as to the mode of life is of importance (luxurious habits, deficient or insufficient nutrition).

Alcoholic and venereal excesses, excessive use of tobacco, addiction to morphin and to cocain should be investigated.

NOTE.—It is not sufficient to ask how much beer, wine, etc., is being taken at the time of examination, but how much had been taken previously. In this way reliable information will be gained.

It is of the utmost importance to make inquiry as to the occupation (as to whether there is sufficient time for recreation, whether the work is regular or irregular, whether attended with mental or physical strain—as at school).

Some pursuits occasion direct injury to health: pulmonary diseases (bronchitis, emphysema) in millers, bakers, stone-masons, workers in factories, or sawmills, or iron-foundries. Intoxications with lead, zinc, arsenic, and mercury occur among plasterers, painters, compositors, and workers in mills. Beer-brewers suffer from diseases of heart, kidneys, nervous system, and liver in consequence of the excessive ingestion of beer (from sixteen to twenty quarts a day).

The situation of dwelling-houses is of significance in relation to some endemic diseases (malaria, articular rheumatism), as well as the contemporaneous occurrence of infectious disease in the neighborhood of a patient suffering from epidemic disease, such as typhoid fever, meningitis, diphtheria, scarlet fever, small-pox, cholera, etc.

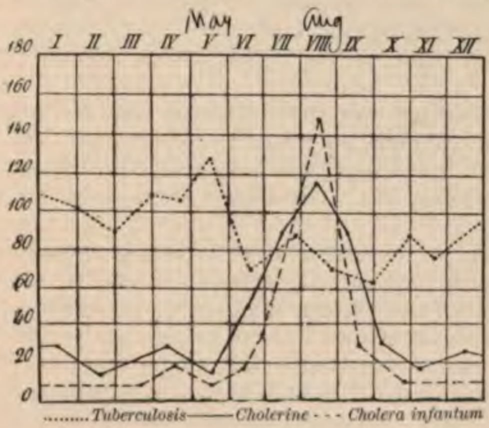

FIG. 1.—Mortality-curve of a large city according to months; shows increase in the mortality from tuberculosis during May, and from cholerine (cholera morbus) in August.

Some diseases are related to climate and season of the year (dysentery, cholera morbus, etc.).

It is important, further, to make inquiry with regard to the previous state of health, and to diseases already recovered from. With regard to the first, especial importance attaches to the periods of infancy and childhood and puberty (advent of menstruation, etc.); and whether or not the constitutional state is a feeble or a robust one. Further, attention is directed to the appearance, the complexion, the mental attitude.

Among previous diseases the infectious diseases are of especial importance, on account of the possible occurrence of sequelæ; thus diphtheria (sequelæ: nephritis, heart-disease, paralysis), scarlet fever (nephritis, suppuration of the middle ear), acute articular rheumatism (heart-disease, chorea), whooping-cough (tuberculosis). Tuberculous suppuration (of glands, bones, and joints) and syphilitic exanthemata are of the greatest significance.

Further, previously frequently repeated attacks of colic (gall-stones, renal calculi), hematemesis (gastric ulcer), hemoptysis (diseases of lungs and heart), frequent catarrhal states (tuberculosis, emphysema), pleurisy (tuberculosis).

Human beings are susceptible to repeated attacks of pneumonia, angina, polyarthritis, erysipelas. As a rule, they suffer from one attack only of typhoid fever, scarlet fever, measles, etc. (Recovery from infection confers immunity.)

Concerning the origin of the diseases named, it is important to inquire into the causes assigned by the patient as operative (traumatism, cold, strain, errors in diet, etc.). Then the mode of onset is to be accurately determined: whether acute (especially infectious diseases, such as pneumonia, malaria, scarlet fever, erysipelas); subacute (typhoid fever, whooping-cough); or chronic (tuberculosis).

Inquiry is next made as to the further course of the disease, and concerning especially prominent symptoms, and finally a mode of treatment is decided upon. To these ends investigation is first directed to the following train of constitutional symptoms:

Bodily Vigor.—Can the patient yet walk, and how far; or is he bed-ridden, and for how long.

A pronounced sense of weakness accompanies progressive wasting diseases, such as carcinoma, pulmonary tuberculosis, chronic nephritis, leukemia, severe diabetes; also febrile states and anemia, chlorosis, neurasthenia, and chronic diseases of the gastro-intestinal tract.

Emaciation.—Former body-weight; appearance; complexion (cachectic appearance associated with carcinoma).

Appetite.—Impaired in febrile disorders, gastric catarrh, carcinoma, etc.; markedly increased in diabetes and during convalescence (typhoid).

Thirst.—Increased in febrile states, after sweating and diarrhea, in diabetes.

Sleep.—Poor in febrile states, in association with pain, with nervous unrest and overactivity; great desire for sleep in chlorosis, uremia, and neurasthenia.

Febrile Symptoms.—Feeling of heat or of chilliness, thirst, headache.

Chill attends malaria, pyemia, onset of pneumonia, ulcerative endocarditis, etc.

Tendency to *sweating*. Night-sweats attend pulmonary tuberculosis.

Especial inquiry is then directed to all the various parts of the body, beginning with the head.

Head.—Pain, vertigo (attending nervous disorders, anemia, ocular disorders, diseases of the ear), derangements of the special senses, coryza, difficulty in deglutition, etc.

Throat.—Voice (hoarseness attending catarrh, paralysis, ulceration of the vocal bands).

Chest.—Pain in the side (pleuritic?); cough (catarrh of the air-passages, pneumonia, dry pleurisy, tuberculosis, circulatory disorders—cardiac lesions); breathing (embarrassed in all profound disorders of the respiratory and circulatory organs, and in conjunction with severe anemia); expectoration (amount, color); palpitation of the heart (nervous, in febrile states, in conjunction with diseases of the heart and with exophthalmic goiter).

Abdomen.—Pain, vomiting (acute poisoning, gastritis, gastric ulcer [blood], carcinoma [chocolate-brown], dilatation of the stomach [large amounts], nervous dyspepsia, stenosis of the esophagus [immediately after ingestion of food], obstruction of the bowel [fecal vomiting, tormina], uremia [toxic], initial symptom of many infectious diseases, cerebral diseases [tumor, hydrocephalus, meningitis]); acid eructation (heart-burn) attends hyperacidity, gastric ulcer, neurasthenia, lactic-acid fermentation (gastric catarrh, carcinoma).

The Bowels.—Constipation or diarrhea (intestinal catarrh, cholera, intestinal tuberculosis, etc.).

Flatulence (intestinal atony, corpulence, abnormal fermentative processes in stomach or bowel).

Micturition (pain, amount).

Extremities.—Pain, swelling (venous thrombosis, general circulatory disorders, nephritis, cachexia).

It will be necessary to inquire in regard to all of these points only in exceptional cases. The proper selection will occasion the experienced physician no difficulty. The beginner and the student, however, should adhere to the schematic arrangement. Further, in accordance with the nature of the case, special manifestations will at times require more detailed study.

The statements of patients must generally be received with a certain amount of reserve, especially voluntary expressions of opinion and diagnoses of previous disease. It is best to be guided by the existing symptoms. Exaggerations are, as a rule, readily appreciated.

THE PRESENT STATE.

This refers to both general and special conditions, and includes all that can be learned by objective examination of the patient at the time.

A. General Manifestations.

In this place will be considered those manifestations that are appreciable at a glance, without further investigation.

1. The state of **consciousness** and of **intelligence** (whether the sensorium be clear or clouded). Apathy, sopor, or coma may attend severe febrile diseases (typhoid, meningitis, septicemia, etc.), cerebral diseases, **intoxica-**

tions; delirium and hallucinations also may be present. Is there marked stupidity, or even idiocy? Speech and writing: disorders of articulation, dysarthria; aphasia (motor [ataxic], sensory, agraphia, alexia, optic [mind-blindness]). Memory.

2. **Height and Bodily Development** (proportions).

3. **Nutritive State.**—Emaciation attends wasting diseases: carcinoma, pulmonary tuberculosis, leukemia; severe diseases of the stomach and the esophagus. The skin under these conditions can be raised in large, loose folds.

4. **Bodily Vigor.**—Estimated in ambulant patients by the gait, whether debilitated, bent, or difficult and dragging; in bed-patients by the posture, whether the normal active position, or passive sinking back into the pillows.

5. **Posture.**—In addition to active and passive attitude in bed, attention is to be directed to certain forced positions, such as a constant sitting posture (orthopnea), attending shortness of breath, lying on the side (in conjunction with unilateral diseases of lungs and pleuræ).

In febrile and delirious states, etc., there may be, among other things, constant throwing of the body to and fro (jactitation).

6. **Constitution and Habitus.**—Tuberculous habitus: thin, emaciated neck, small chest, pallid complexion. Apoplectic habitus: reddened face, thick neck, barrel-shaped chest, obesity.

7. **Skeleton** (slender or robust).—Deformities of the thorax: 1·kyphosis, 2 scoliosis, 3 lordosis—posterior, lateral, or anterior curvature of the spine; kyphoscoliosis is most frequent.

8. **Musculature** (firm, hard, or flabby).—Condition of the muscles (noteworthy atrophy of the extremities, of the muscles of the chest, etc.).

9. **The Skin and the Subcutaneous Connective Tissue.**

a. **Color.**—Abnormal pallor (anemia, internal hemorrhage); redness (fever, excitement, apoplexy); yellowish

·[Humpbacked] 2·[curved] & at curve, convex
-[bend in] convex in

or brownish discoloration (jaundice from occlusion of the biliary passages and in conjunction with septic processes); bronzing (Addison's disease); grayish discoloration (argyria, deposit of silver); cyanosis (bluish-red discoloration of lips, cheeks, nails, etc.), accompanying insufficient absorption of oxygen into the blood, with simultaneous retention of carbon dioxid (circulatory disorders, pulmonary disease).

b. **Exanthemata.**—A number of cutaneous eruptions are of great diagnostic significance.

Measles: coarse papules in crescentic arrangement, sometimes confluent; scarlet fever: at first punctate, later confluent; the roseola of typhoid fever: small, round red spots on the trunk; herpes attending pneumonia, malaria, cerebrospinal meningitis: groups of vesicles on the lips, nose, etc. (wanting in typhoid fever and tuberculous meningitis); miliaria (sweating-sickness): small, clear vesicles, without significance; drug-exanthems: erythema following the use of antipyrin, quinin, etc., acne following the use of iodids and bromids; chicken-pox: papules transformed into vesicles; small-pox: papules progressively increasing in size, changing into vesicles, and these into pustules; various syphilitic cutaneous eruptions (macular, papular, pustular syphilid); the effects of scratching (scabies, pruritus, icterus, etc.).

c. **Abnormal Sweating**, especially at the period of defervescence in pneumonia (critical sweat), malaria, septicemia, etc.; night-sweats (tuberculosis); cold sweats (colic, death-agony, etc.).

d. **Hemorrhages** (petechiæ)—purpura hæmorrhagica, scorbutus, septicemia, severe scarlet fever, etc.

e. **Cicatrices.**—Of especial importance are those secondary to syphilis, the glandular cicatrices of the neck (tuberculosis), the deeply retracted cicatrices of previous caries, and those of small-pox.

f. **Edema.**—Swelling of the skin through collection of fluid in the subcutaneous connective tissue occurs in conjunction with inflammations (painful, redness of the skin), with acute nephritis (face first affected); with venous stasis, either local (phlebitis, thrombosis), or general (derangements of cardiac compensation, cachexia, marasmus, hydremic edema); finally angioneurotic edema.

g. **Cutaneous Emphysema** (entrance of air into the subcutaneous connective tissue: swelling, crepitation on palpation) follows external injuries, and injuries and ulceration of the lungs (through the mediastinum), the larynx, the intestines.

10. **The Bodily Temperature.**—Normal, between 36.5° and 37.5° C. (97°–99.5° F.). Lower temperatures attend collapse, during critical defervescence; higher temperatures, fever, also heatstroke and sunstroke.

Febrile Symptoms.—Reddened face, dry lips, tremulous tongue, hot, burning skin, marked thirst, delirium, sweating, chill, accelerated pulse.

The following distinctions are made:

Subfebrile temperature (from 37.5° to 38° C.—99.5°–100.4° F.).

Slight fever (from 38° to 38.5° C.—100.4°–101.3° F.).

Moderate fever (from 38.5° to 39.5° C.—101.3°–103.1° F.).

High fever (up to 40.5° C.—104.9° F.).

Hyperpyretic temperature (above 41.5° C.—106.7° F.).

After determining the existence of fever, inquiry is made into its mode of origin. It may set in abruptly with a chill (acute disease), or its onset may be insidious (tuberculosis). Next the type of fever is to be established:

Continued fever (the temperature pursues approximately the same level, at most varying 1° during the day), observed especially in typhoid fever.

Remittent fever (daily variations of more than 1°). The remission generally takes place in the morning; an inverse relation is sometimes noted, especially in tuberculosis (hectic fever).

Intermittent fever (afebrile intervals of varying duration), noted especially in pyemia and malaria. (See charts.)

The course of the fever is best studied by means of appropriate charts. In many infectious diseases the course of the fever is typical, the initial stage (rise of temperature) being followed by the fastigium (height of fever), and this in turn by the stage of defervescence (decline of temperature), which may take place by crisis

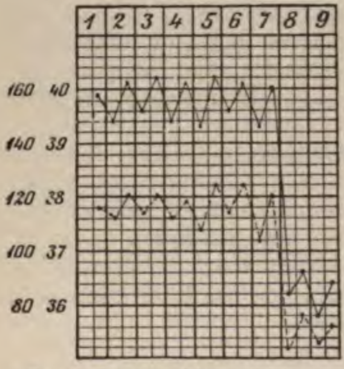

FIG. 2.—Continued fever; crisis on the seventh day of an attack of croupous pneumonia.

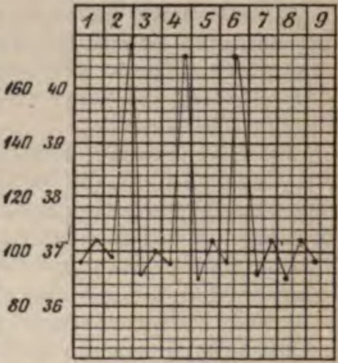

FIG. 4.—Intermittent malarial fever (tertian type).

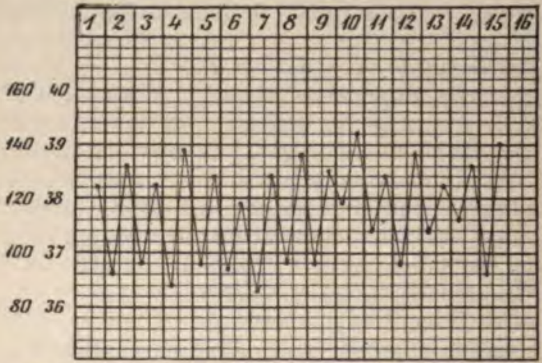

FIG. 3.—Remittent fever (hectic) of chronic tuberculosis.

(in the course of several hours, with perspiration), or by lysis (in the course of several days).

11. In addition to the temperature, attention is directed to the **pulse.** Of importance are the *frequency* (normal 72 in adults, above ~~90~~ per minute in febrile states), the *strength* (strong or weak), the *tension* (soft pulse of fever), and the *rhythm* (irregular in conjunction with disease of the myocardium). For further details see Section III., p. 67, *et seq.*

B. Special Conditions.

The examination of the internal organs is best effected by beginning at the head and progressing downward:

1. **Head.**—The most important points are the size of the skull (hydrocephalus), its shape, and its symmetry.

Eyes.—Position, movements, pupils, color of the conjunctivæ (icteric); conjunctivitis (measles); edema of the lids (acute nephritis). More thorough examination with the ophthalmoscope may disclose retinitis (chronic nephritis), retinal hemorrhages (anemia), retinal infiltrations (leukemia), papillitis (disease of the brain), tubercles in the choroid (miliary tuberculosis).

Nose.—Expansion of the nasal alæ (dyspnea, febrile states in children, pulmonary diseases); shape of the nose (syphilitic saddle-nose); perforations of the septum (tertiary syphilis). Rhinoscopic examination is important in connection with asthma (hypertrophy of the turbinate bodies, polypi), and with hyperplasia of the lymphoid elements (adenoid vegetations) in the nasopharynx (in children with obstructed nasal breathing).

Mouth.

Lips.—Cyanosis, dryness (fever), herpes, anemia.

Tongue.—Mobility; moist or dry, fissured, leathery (typhoid); strawberry-tongue (scarlet fever), coated (gastric catarrh, deficiency of hydrochloric acid), tremulous, smooth (chronic alcoholism).

Teeth.—Carious teeth interfere with normal gastric digestion when mastication is rendered difficult.

Buccal Mucous Membrane; Gums.—Inflammations (stomatitis, gingivitis) attend scorbutus, aphtha, mercurial poisoning; thrush-deposit in children (grayish-white masses of fungi).

Palate.—Inflammation and swelling; abscess-formation; ulceration (syphilis).

Tonsils.—Simple inflammation and swelling (scarlet fever, catarrhal angina), with multiple purulent plugs (lacunar angina), with purulent deposit (necrotic angina), with diphtheric deposit (diphtheric angina); abscess-formation; calculus-formation.

Pharynx. — Catarrhal disorders; chronic catarrh (smooth, atrophic, glazed mucous membrane), in alcoholics, etc.; ulceration (syphilis); retropharyngeal abscess (protuberance).

2. Neck.

Shape: long, thin; short, thick neck.

Depressions.—Abnormal retraction of one supraclavicular fossa in connection with unilateral pulmonary tuberculosis.

Respiratory Muscles.—Striking prominence of the sterno-cleido-mastoid muscles in association with dyspnea, emphysema, etc.

Pulsation of the Jugular Vein.—Diastolic in connection with simple venous stasis; or systolic, true venous pulse of tricuspid insufficiency.

Glandular Swellings; Glandular Cicatrices (tuberculosis); swelling of the cervical glands (syphilis). Post. Ant

Thyroid Gland.—Struma, in association with exophthalmic goiter.

Larynx.—Stenosis (inspiratory dyspnea, laborious, whistling respiration, stridor) in connection with croup, cicatricial contraction, tumors; laryngeal cough (barking) in association with croup, etc. Voice: hoarseness in connection with catarrh, paralysis of vocal bands, tumors, tuberculosis. Further details will be learned through laryngoscopic examination.

Esophagus.—Stenosis, as determined with the aid of the probe; determination of the level of the stenosis (bifurcation of trachea, or cardia); diverticula; auscultation of the sounds generated on deglutition.

3. **Thorax.**

Shape.—Emphysematous (deep, short, barrel-shape); paralytic (long, small, shallow); scoliotic-kyphotic, in connection with deformities of the spinal column; rachitic projections of the sternum (pectus carinatum—chicken-breast); lateral compression of the ribs; funnel-shaped chest; and cobbler's chest (depressed sternum, usually congenital, more rarely acquired).

Symmetry.—Flattening of one side in conjunction with contraction of the lung (chronic tuberculosis, pneumonia) and with pleuritic contraction. Fulness of one side of the chest results from the presence of tumors, pleuritic effusion, and pneumothorax.

Mensuration.—Circumference at the level of the nipple in expiratory position, normally 82 cm. (32¼ in.); in inspiratory position, 90 cm. (35¼ in.). 3

Epigastric (costal-arch) *angle* – *R*, right angle; > *R*, obtuse angle (emphysema); < *R*, acute angle (paralytic thorax).

Respiration.—Symmetric in time; or one side lags behind the other (tuberculosis, pleurisy, pneumonia). *Type.*—Costal (women), costo-abdominal (men); easy, or requiring effort, accelerated, dyspneic. *Dyspnea.*—Inspiratory in conjunction with stenosis of the upper air-passages; especially expiratory in connection with asthma, emphysema, bronchitis; mixed in connection with infiltration of the lungs, compression, circulatory disorders, fever, painful breathing, meteorism of high degree, paralysis of the diaphragm; inspiratory retraction of the thorax (especially in connection with the dyspnea of children).

Cheyne-Stokes breathing: gradual resumption after a pause. *Biot's breathing:* with intervals of varying length.

Pulsation.—Apex-beat (see aneurismal pulsation—either side of the upper portion of the sternum); eventually together with protrusion in connection with aneurism of the aorta.

Lungs.—*a. Percussion.*—Comparison of the qualities of the note on both sides, anteriorly and posteriorly; determination of the limits of the lungs and their motility.

Statement of areas of abnormal dulness, of areas of abnormal tympanitic resonance; precise determination of the limits of such areas (with relation to ribs and spinous processes).

b. Auscultation.—Comparison of the breath-sounds on both sides with regard to character, relation of expiration and inspiration, intensity, and adventitious sounds.

Auscultation of the voice. Study of vocal resonance and of succussion-sounds.

c. Eventual *exploratory puncture* of the pleural cavity.

Heart.—*Inspection and Palpation.*—Abnormal prominences (precordial projection); situation of the apex-beat (within or without the mammillary line); estimated vigor and resistance of the heart-beat.

The presence of epigastric pulsation, visible or only palpable.

Percussion.—Determination of the upper right and left limits. Absolute and relative dulness.

Auscultation of the Four Orifices.—Observation as to vigor, purity, rhythm, and accentuation of the sounds. Murmurs according to character, localization, relation to systole and diastole.

Pulse.—Accurate determination of frequency, rhythm, size, fulness, tension, celerity. Comparison of the pulse upon both sides.

Vessels.—Arteriosclerotic changes (calcareous deposits, tortuosity of the arteries), abnormal pulsation, capillary pulse.

Sounds and murmurs over the large and small arteries, venous hum.

4. **Abdomen.**

Form.—Distended (gas, ascites, tumors, peritonitis), retracted (meningitis).

Symmetry (when tumors are present, usually asymmetric).

Palpation.—Tension of the abdominal walls (in conjunction with pain, peritonitis, and meteorism); superficies, limits, and mobility of tumors possibly present, of the liver; palpation of the spleen and of the usual sites of hernia.

Generation of splashing sounds (dilatation of the stomach), detection of fluctuation (ascites).

Measurement of the abdomen at the level of the umbilicus.

Percussion.—*a*. Of the liver; determination of the lower border; situation and extent of the gall-bladder.

b. Of the spleen, upper and lower anterior boundaries.

c. Of the kidneys.

d. Of tumors possibly present, of a distended bladder, etc.

e. Of ascites possibly present; accurate determination of upper limit; alterations with change in posture.

Exploratory puncture in the case of cysts, ascites, perityphlitis.

Probing the stomach, possibly distention, irrigation, etc.

Examination of the rectum, of the genito-urinary organs.

5. The Extremities.

The lymphatic glands of the axillary, inguinal, and other regions. Alterations in joints and bones.

Drum-stick (clubbed) fingers (chronic disease of the lungs, congenital disease of the heart).

State of motility, of sensibility, of the reflexes, coördination, gait.

6. The Sputum.

As regards amount, separation into layers on standing, color, admixtures. Microscopy (unstained and stained preparations); bacteria.

It may be necessary to examine microscopically and chemically also the nasal mucus, the saliva, and gingival deposits.

7. **The Gastric Contents.**—(Vomited matters or those obtained by siphonage.) Admixture with blood. Microscopy and chemistry (determination of acidity).

8. **The Feces** (admixture of blood, of pus, of mucus). Microscopy. Parasites.

9. **The Urine.**—Amount, specific gravity, color, chemistry (albumin, sugar, etc.), microscopy.

10. **Blood.**—Microscopy. Examination of fresh and stained preparations. Enumeration.

11. **Examination of Eye, Ear, Larynx, and Nares.**

Sphygmography and other scientific precise methods of investigation.

SUBJECTIVE SYMPTOMS.

Pain of all kinds is usually the first manifestation mentioned by patients seeking medical advice. As suspicion will, in consequence, thus be directed in certain directions, some brief consideration will be given to this symptom. The diagnostic significance of subjective symptoms is naturally very variable. Under certain circumstances they may lead at once to diagnostic conclusions (for instance, certain forms of colicky pain), and even apparently slight symptoms should never be ignored as entirely without significance.

It is important to inquire into the *character* of the pain. This may be: inflammatory (throbbing), nervous (shooting), rheumatic (fugacious, wandering).

Syphilitic pains occur especially at night, and are of the character of boring bone-pains.

Colicky pain is spasmodic and very intense. The pain may occur paroxysmally (nervous pain), and recur periodically (neuralgia).

Intoxications of all sorts (alcohol, nicotin, morphin, etc.), as well as auto-intoxications (nephritis, gout, diabetes, gastro-intestinal disorders), are attended with attacks of pain with especial frequency.

The *localization* of the pain is of considerable significance. *Headache* is an important symptom of tumor of the brain (in association with papillitis and vomiting), of meningitis (with rigidity of the neck, fever, etc.), of neuralgia (radiating in the area of distribution of various nerve-branches, as, for instance, the supraorbital, the occipitalis major, etc.), of neurasthenia (habitual headache, heredity), migraine (unilateral, vomiting, flittering scotoma), of anemia and chlorosis (toxic ?), of infectious diseases (especially typhoid fever, beginning tuberculosis, etc.), of intoxications (uremia attending nephritis, nicotin-poisoning, etc.), of diseases of the eye (muscular insufficiencies, iritis, etc.), of the ear (catarrh of the middle ear, etc.), of the nose and its accessory cavities (catarrh, empyema, polyps, tumors).

When *pain in the face* is present there are to be thought of neuralgia (infraorbital, inframaxillary, alveolar), diseases of the nasal cavities, and diseases of the teeth.

Difficulty in deglutition suggests inflammatory conditions of the tonsils, of the palate, abscess-formation, ulcerative processes in the pharynx (syphilis) or the epiglottis (tuberculosis), diseases of the esophagus (stricture).

The most frequent variety of *pain involving the trunk* is muscular pain (rheumatism, the strain of coughing, involving the diaphragm and the muscles of neck, chest, and abdomen).

Neuralgia (intercostal, lumbar, herpes zoster).

Pleuritic, Pericarditic, Peritonitic Pains.—Typically, the pain of perityphlitis is usually localized in the cecal (right iliac) region. The pain of angina pectoris is sharp, vice-like, in the region of the heart, and often also in the arms.

Colicky Pains.—Cardialgia, spasm of the stomach (neurasthenia, gastric ulcer, dilatation, etc.), in the epigastrium ; gall-stone colic, radiating from the liver ; renal colic, pain in the course of the ureter, also attending wandering kidney ; intestinal colic (intestinal catarrh, incarceration of the bowel) ; lead-colic (plumbism), severe abdominal pains with constipation.

Attacks of ill-defined colicky pain should suggest the crises of locomotor ataxia, or girdle-sense.

Pain in micturition attends gonorrhea (especially before and during the act), cystitis (especially after the act), the passage of calculi or gravel, hypertrophy of the prostate, and strictures.

Pain attends evacuation of the bowels in connection with spasm of the sphincter (tenesmus), in consequence of severe diarrhea or of ulceration; with fissures of the anus and fistulæ; with hemorrhoids; with obstinate constipation.

Pains in the extremities are most commonly of the following varieties: rheumatic muscular pains, pains induced through traumatism (contusions, etc.); further inflammatory pains (phlegmons, tenovaginitis, phlebitis); in consequence of osseous deformities (curvatures of the spinal column, flat-foot); gouty pains (podagra, chiragra); neuralgic pains (sciatica); pains dependent upon circulatory disorders, such as phlebectasia (leg-ulcer), phlebitis, arteriosclerosis (gangrene, asphyxia).

SECTION II.

GENERAL CONSIDERATIONS UPON METHODS OF INVESTIGATION.

The various methods of investigation that have been developed in the course of time are all based upon employment of the five senses, with the aid of which, supplemented in numerous ways by instruments and reagents, successful inquiry can be made into the nature of existing morbid processes. In applying these methods clear notions must be had as to their limitations and reliability, and the significance of the results obtained is to be modified accordingly.

The methods of investigation may be classified as follows:

- *A.* Inspection.
  - I. Immediate inspection of
    - Form;
    - Color;
    - Position;
    - Mobility.
  - II. Mediate inspection:
    - 1. With instruments:
    - *a.* Microscopy of—
      - *α.* Fresh preparations;
      - *β.* Stained streak-preparations and sections;
      - *γ.* Enumeration.
    - *b.* Mirror examinations:
      - *α.* Ophthalmoscopy;
      - *β.* Laryngoscopy;
      - *γ.* Otoscopy, rhinoscopy;
      - *δ.* Cystoscopy, gastroscopy (endoscopy).
    - *c.* Thermometry.
    - *d.* Mensuration with tape, compasses, cyrtometer.
    - *e.* Spirometry (measurement of the pulmonary capacity).
    - *f.* Graphic methods:
      - *α.* Sphygmography;
      - *β.* Cardiography, etc.

A. Inspection.

- II. Mediate inspection.
  - *g.* Exploratory puncture;
  - *h.* Bacteriologic methods, etc.
  - 2. With reagents:
    - Chemic investigation (qualitative and quantitative) of—
      - *a.* Blood;
      - *b.* Urine;
      - *c.* Sputum;
      - *d.* Gastric and intestinal contents, etc.

B. Palpation.

- I. Without instruments:
  - Palpation of the surface of the body and the accessory cavities (touch).
- II. With instruments:
  - Examination with the sound of—
    - 1. Esophagus and stomach;
    - 2. Urethra and bladder;
    - 3. Nose, larynx, etc.

C. Examination through the sense of hearing:

- I. Percussion: examination of the internal organs with regard to their air-content.
  - 1. Immediate percussion with the finger;
  - 2. Mediate percussion (finger-finger, finger-pleximeter, plexor-pleximeter).
- II. Auscultation: examination of murmurs generated within the interior of the body,
  - 1. Immediate;
  - 2. Through the mediation of the stethoscope.

D. The sense of smell may be employed in examination of the urine, of the sputum (offensive expectoration), pus (putrid), as well as in the application of certain chemic methods.

The application of the most important methods in general and the instruments necessary therefor, in so far as these are of significance for the practising physician, will now be briefly considered in the order tabulated. Special details are to be looked for in Section III.

1. Microscopy.

Instruments Required:

Microscope (with at least two objectives, one for high and one for low power, 80–500); medium ocular, stand with rack and pinion, revolving iris-diaphragm, illuminating apparatus. Immersion-systems are not necessary, though desirable.

Platinum needle, pipets, watch-glasses.

Slides (English form), cover-glasses.

Reagents: 0.6 per cent. sodium-chlorid solution (indifferent); 1 per cent. acetic-acid solution (for clearing albuminous bodies); glycerin (for clearing and preservation); osmic acid (0.5 per cent.), stains fat black; alkalies (3 per cent.), for elastic fibers; formalin (10 per cent.), a good hardening-agent—*e. g.*, for blood-preparations; Lugol's solution of iodin (yellowish-brown stain); eosin-solution and Bismarck-brown (1 per cent.), stain cells diffusely; hematoxylin and methylene-blue stain the nuclei; fuchsin and gentian-violet (1 per cent.) are most commonly used to stain bacteria. Good double stains are obtained by means of solutions of hematoxylin and eosin, and methylene-blue and eosin.[1]

For imbedding specimens Canada balsam (dissolved in xylol) is used.

Unstained Preparations.

For the study of unstained preparations the smallest amount possible of the material to be examined is used, and this is spread upon a slide in a thin layer by means of a platinum needle previously sterilized (by heating in a flame). Further, reagents, such as sodium chlorid, acetic acid, solution of iodin, osmic acid, may be added. A cover-glass is applied firmly to the slide in order to expel air-bubbles. In viewing unstained preparations through the microscope the light should not be too strong.

Stained Preparations.

After the material to be examined has been spread in a thin layer upon the cover-glass the preparation is permitted to dry in the air, without the application of heat, for fifteen minutes and more. It is then passed slowly three times (in the case of blood eight or ten times) through the flame of a Bunsen burner or of a spirit-lamp in order to fix the structures. Next several drops of the staining-solution are placed upon the spread preparation and the cover-glass is warmed until the vapor of steam escapes (one minute usually suffices); then the cover-slip is washed in water, the excess of which is removed with bibulous paper, and dried in the air, with final enclosure in Canada balsam. The staining can be effected also in a watch-glass containing the stain, with the application of heat.

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[1] The best stains are those of Gruebler, of Leipsic. The solution of hematoxylin and eosin is constituted as follows:

Hematoxylin, 5.;
Acetic acid, 20.;
Distilled water, alcohol, glycerin, of each, 100.;
Alum in excess;
Eosin, 0.5.

To be filtered whenever used. Duration of staining, ten to twenty hours.

The most convenient staining-procedure, which is sufficient for preliminary examination in most cases, is that with the solution of methylene-blue and eosin.[1] The nuclei of the cells and bacteria appear blue, the bodies of the cells and the red blood-corpuscles red. In unstained preparations structure is shown better, and fat-drops remain unchanged, but not in stained preparations, in which, however, the nuclei of the cells and bacteria appear more conspicuously. Stained preparations, further, may be preserved, while pencil-drawings of fresh preparations should always be made and systematically preserved, together with notes of date, name, and other conditions.

Sediments.

Solid substances found suspended in considerable fluid, such as urine, exudates, etc., are permitted to settle in conical glasses, and in the course of from three to ten hours the sediment can be sucked up by means of a pipet. The sediment can be obtained to better advantage and more speedily by means of centrifugation.

2. Examination with Mirrors.

For the successful examination of the cavities of the body with the aid of mirrors it is desirable to have a suitable space which can be enclosed in black cloth.

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[1] The solution of methylene-blue and eosin is constituted of—
Concentrated aqueous solution of methylene-blue, 60.;
½ per cent. solution of eosin in 70 per cent. alcohol, 20.;
Distilled water, 40.;
20 per cent. solution of potassic hydrate, 12 drops.

A good petroleum-lamp with a circular wick and cylindric chimney will answer as a source of light.

By means of a reflector the light is thrown into the cavity to be examined—*e. g.*, the eyeball, the mouth, etc.

In making laryngoscopic examinations the reflector is fastened upon the forehead. A small laryngoscopic mirror, warmed in order to avoid deposition of vapor, is slowly introduced, while the extended tongue is held firmly. The patient is made to say "hae" in inspiration. After a little experience examinations of this kind are readily made.

In examination of the nose and ear suitable specula are employed, and the light is projected through these. By varying the position of the reflector all portions of these cavities can be successively illuminated.

Concerning details of the technic the larger text-books must be consulted.

3. Thermometry.

In studying the temperature of the body clinical (self-registering) thermometers are employed, the column of mercury remaining at the level to which it was elevated, and being shaken down by a sudden, vigorous jerk before being used again.

The temperature may be taken in the axilla, in the rectum, in the mouth, or in the fold of the groin. The instrument should be permitted to remain *in situ* for from three to ten minutes, and when applied to the external skin all moisture should previously have been removed. The exposure in these situations should also be longer than within the mucous cavities.

The axillary temperature is about one degree lower than that of the rectum, and about half a degree lower than that of the mouth.

The temperature is, as a rule, taken morning and evening, and systematically recorded upon appropriate charts.

4. Exploratory Puncture.

The object of exploratory puncture is to convey information with regard to the presence of deep-seated accumulations of fluid, pus, blood, etc. The procedure is to be employed only when the diagnosis cannot be reached by means of other methods. Properly practised, it is entirely without danger. For this purpose so-called exploratory syringes are employed, which are provided with longer and stronger needles than ordinary hypodermic syringes. The needles are sterilized by boiling both before and after being used, and are carefully dried before being put away.

Puncture is usually made in the situation of most marked dulness, the overlying skin being previously cleansed most carefully and then washed with alcohol and 3 per cent. solution of carbolic acid. In introducing the needle the resistance of the penetrated layers should be noted (thick cicatrices, abscess-membranes). Should fluid not appear immediately, the needle is cautiously introduced more deeply or is slightly withdrawn and the attempt repeated. The opening of puncture may be closed with aseptic collodion or a bit of adhesive plaster.

Exploratory puncture is most frequently practised in cases of exudative pleurisy (to determine whether the exudation is serous, purulent, or hemorrhagic), of exudative pericarditis, of abdominal tumors (cysts, hydronephrosis), of abscess-formation (perityphlitis).

Of late, it has become customary to practise exploratory puncture of the spinal membranes in the lumbar region in cases of meningitis, hydrocephalus, and hemorrhage, to determine the nature of the contents of the subdural lymph-space.

5. Bacteriologic Methods of Investigation.

a. Staining.—For the preparation of cover-glass specimens, see p. 21.

All bacteria stain well with basic aniline dyes (methylene-blue, gentian-violet, fuchsin, Bismarck-brown). It is best to have these stains in concentrated stock-solution, which may be appropriately diluted as required (five drops to one-tenth of a test-tube of water).

Methylene-blue and Bismarck-brown stain more slowly, while fuchsin, and especially gentian-violet, readily overstain. Especially useful staining-solutions are Loeffler's alkaline methylene-blue solution[1] and Ziehl's carbol-fuchsin solution.[2]

For double staining the solution of methylene-blue and eosin (see p. 22) may be employed.

For the isolated staining of bacteria Gram's method may be pursued, though not universally applicable. For this are required: fresh gentian-violet-aniline-water solution (prepared by adding ten drops of aniline oil to one-fourth of a test-tube of distilled water, agitating the mixture, filtering, and adding to the filtrate five drops of a concentrated solution of gentian-violet. With this solution the cover-glass preparation is stained without heat for one minute, and when dry is placed in Lugol's solution of iodin for one minute. It is then dried and exposed to the influence of absolute ① alcohol for ten minutes. After being washed in water it is stained with eosin or with Bismarck-brown. The bacteria appear blue and all other structures red or brown.

Fixes
in B
Decol.
[illegible]
bact.
[illegible]

This stain is applicable to staphylococci, streptococci, pneumococci, tetanus-bacilli, anthrax-bacilli, tubercle-bacilli, but not to cholera-bacilli, typhoid-bacilli, influenza-bacilli, spirilla of relapsing fever, gonococci.

For tubercle-bacilli there is a specific stain, as these organisms retain the stain more tenaciously than other microörganisms, even when treated with acids. The most convenient and the most reliable method is as follows: The dry cover-slip preparation is stained for fifteen minutes in a warm carbol-fuchsin solution. It is then decolorized for several seconds in a 5 per cent. solution of sulphuric acid. After being washed with water the preparation is counterstained with methylene-blue. It is now washed again, then dried slowly in the air or more rapidly over the flame of a Bunsen burner, and finally imbedded in Canada balsam.

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[1] Concentrated alcoholic solution of methylene-blue, 30.;
Potassium hydroxid (0.01 per cent.), 100.;
Loeffler's solution of methylene-blue.

[2] Fuchsin, 1.;
Alcohol, 10.;
Liquefied carbolic acid, 5.;
Distilled water, 100.;
Carbol-fuchsin.

When but small numbers of the bacilli are present the following mode of procedure may be pursued: A half test-tubeful of pus, sputum, etc., is boiled for fifteen minutes over a water-bath. Preparations are then made from the resulting precipitate, which contains coagulated albuminous bodies and tubercle-bacilli (Dahmen's procedure).

b. Cultivation.—For the clinician the demonstration of cholera-bacilli, anthrax-bacilli, diphtheria-bacilli, pyogenic microörganisms, by culture-methods is of importance, and under certain circumstances also that of typhoid-bacilli and gonococci. To these ends test-tubes containing solid culture-media are kept in readiness, nutrient agar-agar (which retains its firmness at the temperature of the body), and nutrient gelatin (which liquefies at this temperature), being perhaps the most available.

There will be further required various shallow dishes, empty test-tubes, etc., sterilized by dry heat at a temperature of 160° C. (320° F.).

Anthrax-bacilli; Pyogenic Microörganisms.—Anthrax-matters and purulent exudates are inoculated by stroking a previously heated platinum needle upon the surface of hardened agar or gelatin. In a warm temperature there develop within twenty-four hours colonies, which are studied further microscopically in cover-glass preparations. The characteristic colonies of these respective microörganisms are then inoculated upon new tubes, and in this way pure cultures are obtained.

Anthrax-bacilli form brownish-yellow colonies, with masses of densely interwoven wavy threads; the staphylococcus albus white, the staphylococcus aureus golden-yellow, the staphylococcus citreus round-yellow colonies, the streptococcus pyogenes small grayish points, and likewise the diplococcus pneumoniæ.

Cholera-bacilli.—A small flocculus of the suspected dejection is introduced into a test-tube half-filled with sterile 1 per cent. peptone-solution containing 0.5 per cent. sodium chlorid. At a temperature of 37° C. (98.6° F.) the bacilli appear in positive cases in the course of eight or ten hours upon the surface in pure culture. From time to time microscopic preparations are made. With a drop of the culture inoculations are made upon warmed liquid gelatin, which is then poured into a sterilized dish and permitted to solidify. At a temperature of from 20° to 22° C. (68°–71.6° F.) in the thermostat the colonies appear somewhat depressed in from thirty-six to forty-eight hours (through liquefaction of the gelatin). The colonies present a light-yellow, granular appearance, as if studded with small bits of glass, and being slightly serrated at the margin (with low power).

Diphtheria-bacilli.—Inoculations are made upon glycerin-agar or upon simple agar smeared with human blood, the infective material being spread upon the surface of the culture-medium. In the course of from twelve to sixteen hours, at a temperature of 37° C. (98.6° F.), characteristic colonies resembling bright white drops are developed.

Typhoid-bacilli.—Pure cultures may be obtained by pouring into dishes gelatin inoculated with material from a typhoid stool. In the course of two or three days the colonies are distinctly visible, displaying a light-brownish color and a somewhat irregular border. Cultivated in this way they exhibit, however, too few points of differentiation from other bacteria (for instance, the bacterium coli, various water-bacteria, etc.). Their growth upon boiled sterile potato is quite characteristic, resulting in the development of a very delicate, scarcely visible, moist deposit. Further, typhoid-bacilli do not cause coagulation of sterile milk, and do not induce gas-formation in solutions of grape-sugar, as, for instance, does the bacterium coli.

Of late the Gruber-Widal serum-reaction (agglutination and immobilization of typhoid-bacilli exposed to blood-serum from a case of typhoid fever) has proved to be a valuable means of diagnosis and of identification of typhoid-bacilli. (For further particulars see p. 140.)

c. *Inoculation.*—In doubtful cases the demonstration of the presence of tubercle-bacilli in exudates, etc., may be made by injecting into the abdominal cavity of guinea-pigs or rabbits a hypodermic syringeful of the material secured with antiseptic precautions. In the course of from four to six weeks, in a positive case, tuberculosis of the peritoneum will be distinctly demonstrable. A similar course of procedure may be pursued also for the demonstration of the diplococcus pneumoniæ. Death occurs after two or three days, and in the blood may be found immense numbers of diplococci.

6. Palpation.

Manual palpation is performed with the tips of the fingers, the nails being cut short. Through practice and exercise the sense of touch may be highly developed, a matter of considerable importance in palpation of the abdomen.

Palpation is directed to the study of certain movements, such as the apex-beat of the heart, epigastric pulsation, arterial pulsation, etc., with regard to localization, extent, and strength.

With the hand laid flat vocal fremitus (the vibration of the voice transmitted to the chest-wall) is studied. This may be increased or diminished abnormally in accordance with the state of the conductors of sound (bronchi, lungs). Ab~~normal friction-sou~~nds (pleuritic friction, dry râles) may also be thus felt.

Abnormal enlargement of organs (liver, spleen, etc.) may be felt through the relaxed abdominal walls (quiet breathing, with open mouth, in a comfortable position, the lower extremities being slightly drawn up). Should the results of palpation be uncertain (small tumors, adipose abdominal walls, pain, etc.), the examination should be made under chloroform-narcosis. In the case of tumors especial attention should be directed to mobility with respiration and other movements.

The presence of free fluid in the abdominal cavity (ascites) is determined by the existence of fluctuation, the wave generated by quickly tapping one side of the abdomen (with the hand of a second person placed on edge in the median line, to prevent the transmission of the movement of the abdominal wall) being felt with the hand applied upon the opposite side.

In making examinations with sounds or probes these are first moistened with water, or with oil, to facilitate their introduction.

When a probe is to be introduced into the esophagus the patient should preferably be seated upon a chair with a high back, and be instructed to breathe quietly, the probe being permitted to pass over the fingers of the left hand introduced into the mouth.

7. Percussion.[1]

In accordance with the amount of air present in a body, this will yield upon tapping a special note (full or empty barrel). As, however, the internal viscera contain varying amounts of air, percussion furnishes a means of—

1. Determining the boundaries that separate one from the other organs of varying air-content that lie in close proximity; and
2. Of reaching conclusions as to pathologic states of organs from abnormal variations in the note they yield.

Percussion is either immediate (direct tapping of the surface of the body) or mediate (interposition of a solid body, such as the finger or a pleximeter, in tapping).[2]

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[1] Percussion was discovered by L. Auenbrugger, of Vienna (1761), "Inventum novum ex percussione thoracis, etc.," and its application was further extended by Corvisart. Piorry invented the pleximeter in 1826, and Wintrich the percussion-hammer in 1841.

[2] Immediate auscultation has long been practised (succussio Hippocratis); mediate auscultation was discovered by Laennec (1816, *Traité de l'Auscultation médiate*, etc.), who determined the auscultatory phenomena of most diseases. Skoda subsequently elaborated critically the results of Laennec's observations.

The simplest and best mode of practising percussion consists in the use of the fingers of both hands. The palmar aspect of the middle finger of the left hand is firmly applied to the surface of the body, and receives blows struck with the middle finger of the right hand flexed at a right angle at the first interphalangeal joint. Especial care should be taken that the movement is made from the wrist. This mode of percussion should be practised until a distinct note is obtained upon the thigh. Then resort may be had to percussion with hammer and pleximeter. The determination of the percutory limits of an organ is effected in general through light percussion.

The different qualities of note thus elicitable are—

1. A *dull, low* note, yielded upon percussion of airless bodies, whether fluid or solid; and

2. A *clear, loud* note, yielded upon percussion of organs containing air. higher

Between these two extremes there exist gradations, as with increasing size of the vibrating layer of air the note becomes clearer, while with diminishing size it becomes duller. Under the latter conditions the designation *relative dulness* is employed. When there is a total absence of air the note becomes *absolutely dull.* The clear and the relatively dull note may be either tympanitic (ringing) or not, according as the vibrations follow one another with relative periodicity or not. It is *tympanitic* when the vibrations take place in a space with walls not too tense; but when the tension is great the note is no longer tympanitic.

The tympanitic note may further be *high-pitched* or *low-pitched*, in accordance with the number of vibrations in a unit of time. The smaller the cavity the higher the note.

A variety of the tympanitic note is the *metallic-sound* (depending upon a predominance of higher overtones). This may be heard over cavities with smooth walls. Upon tapping with the stile of the percussion-hammer

tart fr. Dulln. → Resonance

inger = w. border you are after.

upon the applied pleximeter the bell-like note is heard with especial distinctness through a stethoscope.

A dull note is yielded normally by all solid portions of the body (for instance, the thigh, the head, the region of the vertebral column); further by the heart, the liver, the spleen, the kidneys, where these lie in close contact with the walls of the body. In situations in which air-containing organs lie between solid viscera and the walls of the body there is developed a relatively dull note.

The lungs yield a clear but not tympanitic note, while stomach, bowel, larynx, and trachea yield a tympanitic note.

Pathologic dulness develops when solid or liquid substances displace air-containing organs or contained air; a *pathologic clear note* when solid organs are displaced by air; ~~*abnormal tympany*~~ over relaxed lungs and when ~~cavities~~ have formed in the ~~lungs~~.[1]

8. **Auscultation.**

There are generated within the body a variety of sounds of normal or pathologic character, which may be heard in part at some distance (certain râles and splashing-sounds), in part only on applying the ear to the body-wall.

For the distinct and isolated auscultation of these sounds the stethoscope is generally employed as a conductor. The instrument should be applied vertically and firmly, though without undue pressure.

The mechanism of the origin of the murmurs is a variable one. It is referable partly to the vibration of currents of air, partly to the movement of fluid in spaces filled with air, partly to the tension of membranes and muscles, the rubbing of roughened surfaces upon one another, and the like.

The most important sounds thus developed in connection with *respiration* are—

1. The *vesicular respiratory murmur*, which is heard

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[1] The lungs removed from the thorax, and thus freed from tension and relaxed, yield a tympanitic note.

over the normal, breathing lung. It is generated wherever pulmonary alveolar respiration (cellular respiration) takes place. Where this murmur is wanting pathologically the conclusion is justified that there exists derangement of vesicular respiratory function.

Vesicular breathing is distinctly audible only during inspiration and corresponds with a soft *v*.

2. The *bronchial respiratory murmur* (resembling the rushing sound generated on blowing through a tube) is heard normally over the larynx, the trachea, and the bronchi, and under pathologic conditions when vesicular breathing is abolished, and only that of the bronchi is audible. It is louder during expiration than during inspiration and corresponds to the sharp *ch*.

An especial form of ringing breathing is amphoric breathing (corresponding with the sound produced by blowing over the opening of a bottle). This occurs under similar conditions as the metallic percussion-note.

3. *Transmission of the Voice.*—During speech a murmuring sound is heard over the healthy lung, which may be enfeebled or exaggerated pathologically. Enfeeblement occurs in conjunction with obstruction of the bronchi and displacement of the lungs from the chest-wall; exaggeration (bronchophony, pectoriloquy) in connection with condensation of the pulmonary tissue. Egophony (bleating-sound) is noted in connection with incomplete compression of the lung.

4. *Râles* occur when fluid is present in the bronchi, through the bursting of air-bubbles, and when the walls of the smallest bronchial tubes stick together and are suddenly pulled apart. They may be scanty or numerous, dry or moist, ringing or toneless, in accordance with the character of the fluid (viscid, mucous, liquid), and the space within which they are generated (large or small bronchi, cavities).

5. *Pleuritic friction* occurs in conjunction with deposits upon the pleural surfaces. It occurs intermittently and resembles the creaking of leather.

6. *Succussion-sounds* (metallic splashing-sounds) occur in association with seropneumothorax (simultaneous presence of fluid and air in the pleural cavity) when the patient is abruptly shaken from the shoulders.

Murmurs generated in the *circulation* are divisible into those heard over the heart and those heard over the vessels.

Over the healthy heart two sounds can be heard at all of the orifices. Through the sudden tension of the valves there results the aortic and pulmonary second sound (diastolic), as well as the mitral and tricuspid first sound (systolic). To the latter the contraction of the muscular wall of the heart also contributes. Through the systolic tension of the walls of the vessels there results the first sound over the aorta and the pulmonary artery. The second sound over the mitral and tricuspid is transmitted from the aorta and pulmonary artery. These sounds may be stronger (second aortic, second pulmonary, first mitral, first tricuspid) or weaker in accordance with differences in tension. They may be more or less irregular in rhythm in accordance with the synchronousness in the activity of the ventricles and auricles (division, duplication). They may finally be entirely wanting in connection with disturbances of valvular activity, being replaced or separated by murmurs.

The *heart-murmurs* heard in connection with valvular lesions arise from the generation of abnormal currents in the blood-stream, whether due to the forcing onward of blood through narrowed orifices (stenosis) or regurgitation through abnormal orifices (insufficiency). The acoustic transmission of these sounds takes place best in the direction of the blood-stream responsible for their generation.

The murmurs may be blowing, bubbling, purring, rasping, rumbling. It is important to determine the situation at which they are best heard and the period of their occurrence (whether systolic, diastolic, or presystolic).

From these organic murmurs are to be distinguished so-called accidental murmurs, which may occur, in the absence of valvular lesions, in connection with disturbances of myocardial activity and in the rapidity of movement of the blood-stream (anemia, fever, etc.). They are almost exclusively systolic and of a soft, blowing character.

In the presence of deposits upon the pericardial layers there results *pericarditic friction* (intermittent, rough, and apparently closer to the ear than endocarditic friction).

Over the large vessels (subclavian, carotid) the same sounds are audible as over the aorta. Over the smaller vessels normally only upon pressure with the stethoscope a diastolic murmur can be heard, and with increasing pressure a distinct sound.

Abnormal sounds over the vessels occur in connection with aortic insufficiency.

Over the jugular vein in anemic persons a peculiar continuous *hum* may often be heard, which is probably attributable to the increased rapidity of the blood-stream in an imperfectly filled vein.

Of murmurs generated in the digestive tract auscultation may be directed to the study of the *swallowing-sound*, which may be heard at the termination of the esophagus in the epigastrium, or posteriorly to the left of the spinal column, immediately after the act of swallowing, as a short splashing-murmur, and which may be absent under pathologic conditions (stenosis). Anteriorly there may be heard in addition to the first also a second swallowing-sound (secondary murmur), due to the formation and rupture of air-bubbles.

Intestinal murmurs result through peristalsis, in conjunction with the simultaneous presence of gases and fluids, and are abundant with increased peristalsis. Loud *splashing-sounds* develop in the stomach under normal and pathologic conditions (dilatation). When peritonitic deposits form upon the surface of the bowel and the liver *peritonitic friction* or creaking may under some circumstances be heard.

SECTION III.

SPECIAL DIAGNOSIS OF DISEASES OF THE INTERNAL ORGANS.

I. EXAMINATION OF THE RESPIRATORY APPARATUS.

1. Nose and Nasopharyngeal Space.

The *form* of the nose may be modified by defects in the septum: for instance, it may be depressed in the shape of a saddle, as a result of syphilitic disease. At the entrance of the nares eczema is likely to develop in conjunction with catarrhal conditions. If nasal breathing be obstructed (by catarrh or tumors), breathing through the mouth takes its place vicariously (as indicated especially by snoring at night and great dryness of the mouth in the morning). When dyspnea exists the nasal alæ become dilated in consequence of the respiratory effort.

Further examination is made with the aid of a nasal mirror (anterior and posterior rhinoscopy); also by means of sounds or probes.

Anterior rhinoscopy is practised by means of a nasal speculum and a reflecting mirror.

Attention is directed to the position of the septum (deflections may cause difficulty in breathing), to the presence of ulcers (syphilitic perforations, tuberculous ulceration); also to the form of the turbinated bodies (swelling, hypertrophy; nasal asthma may arise from this source reflexly), to the presence of polypi (mucoid-vitreous masses), to abnormal collections of crusts (ozena, in association with fetid odor).

Epistaxis is frequently associated with a circumscribed area of ulceration upon the septum. This occurs especially in connection with chlorosis, profound chronic visceral disease, and leukemia. Suppuration in the accessory cavities (empyema of the frontal sinuses, or of the maxillary sinuses) is demonstrable by puncture.

Posterior rhinoscopy is practised in a similar manner to laryngoscopy, except that smaller pharyngeal mirrors are employed. These are introduced behind the uvula and are illuminated by means of a reflecting mirror, the tongue being held out of the way by means of a depressor.

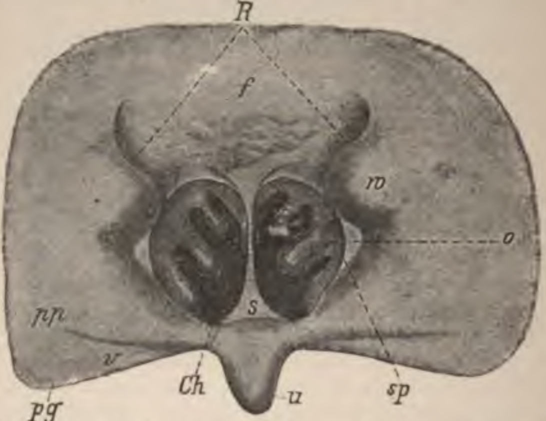

FIG. 5.—Rhinoscopic view of the nasopharyngeal space: *s*, septum; *w*, tubal prominence; *Ch*, choana; *R*, fossa of Rosenmüller; *o*, orifice of tube; *u*, uvula; *pg*, palatoglossal process.

By this means especially may be recognized retronasal tumors, the most frequent of which are adenoid vegetations (hypertrophy of the pharyngeal tonsils in the fossa of Rosenmüller in children), which may also be palpated by means of the finger. (Symptoms: obstructed nasal breathing, constantly open mouth, hardness of hearing, mental deficiency.)

2. Larynx and Trachea.

The diseases of the larynx manifest themselves, in accordance with their character and localization, in disturbances of breathing, of voice-formation, and of the act of deglutition.

In cases of dyspnea dependent upon *stenosis* of the larynx this organ makes wide respiratory excursions and the head is thrown backward; while in cases of stenosis below the larynx (tracheal stenosis) this organ remains still and the head is bent forward. Suffocative attacks occur in connection with tumors, especially when seated upon the vocal bands; with spasm of the muscles of the glottis and with paralysis of the muscles that separate the vocal bands in inspiration (posterior crico-arytenoid).

Laryngeal dyspnea is inspiratory and attended with loud stridor (hissing sound). In addition to the diseases named it occurs most frequently in association with laryngeal croup (narrowing of the lumen of the larynx by deposits of membrane) and with edema of the glottis.

Laryngeal cough is sometimes strikingly loud and barking (croupy cough).

Even slight irritation of the larynx is capable of inducing intense reflex cough.

During normal respiration the chink of the glottis is widened (respiratory glottis) in inspiration through the action of the posterior crico-arytenoid muscles (abductors). It is closed during expiration through the action of the lateral crico-arytenoid and the interarytenoid muscles (adductors). For the production of voice the vocal bands must be rendered tense through the action of the cricothyroid muscles and especially and principally through the action of the thyro-arytenoid.

All of the muscles of the larynx receive their nervous supply through the inferior (recurrent) laryngeal nerve, with the exception of the cricothyroid muscle, which receives its supply through the superior laryngeal nerve. Both nerves are branches of the vagus (accessory nucleus in the lower portion of the medulla oblongata).

If the tension of the vocal bands is not properly effected, hoarseness results (catarrhal swelling, ulcerative processes, paralysis of the vocal bands). Aphonia is the condition in which only whispering voice is possible. A shrill voice (abnormally high and thin) occurs principally in association with functional disorders. Double voice (diplophonia) occurs in connection with some forms of tumor of the vocal bands. When nasal obstruction exists (catarrh, tumors) the voice becomes "stopped up." In failure of the palate to close (ulceration, paralysis) the voice becomes nasal and open.

By means of laryngoscopic examination (for technic see p. 23) a view is obtained of the epiglottis above, the aryepiglottic folds upon the sides, and the interarytenoid region posteriorly between the arytenoid cartilages. Within the space bounded by these parts are seated the white, tendinous true vocal bands and above them the ventricular bands (Figs. 6 and 7).

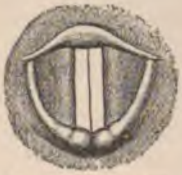

FIG. 6.—Phonation.

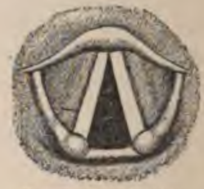

FIG. 7.—Inspiration.

Attention is directed to the movements of the vocal bands during respiration and phonation; also to abnormal redness, swelling, ulceration, tumor-formation.

Redness attends catarrhal conditions (rose vocal bands).

Swelling occurs under like conditions, as well as in conjunction with deeply situated abscesses (perichondritis).

Ulceration (of the vocal bands, of the epiglottis, of the interarytenoid space) is mostly tuberculous (Fig. 8), less commonly syphilitic (with radiating cicatrices).

Tumors.—Polypi seated upon the vocal bands, fibroma, carcinoma of the vocal bands.

Paralysis of the vocal bands: from catarrh, neuritis (postdiphtheric), compression-neuritis, disease of the medullary nuclei (tabes, sclerosis, syringomyelia), in cases of hysteria. The most frequent variety is *paralysis of the thyro-arytenoid muscle* (Fig. 9, oval glottis): aphonia, no dyspnea (attending laryngitis, functional over-activity, hysteria). Next in frequency is *paralysis of the transverse arytenoid muscle* (Fig. 10), which results in a small triangular fissure between the arytenoid cartilages during phonation: hoarseness, no dyspnea (catarrh, hysteria).

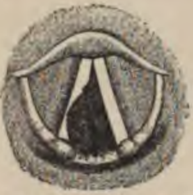

FIG. 8.—Tuberculous laryngitis.

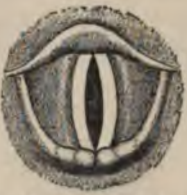

FIG. 9.—Paralysis of the thyro-arytenoids. (Phonation.)

Paralysis of the posterior crico-arytenoid (Fig. 11) is attended with pronounced inspiratory dyspnea if bilateral (failure of the chink of the glottis to open during inspiration), while the voice remains normal (tabes, sclerosis, syringomelia).

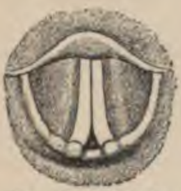

FIG. 10.—Paralysis of the transverse arytenoid. (Phonation.)

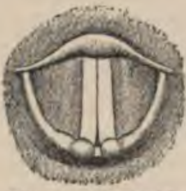

FIG. 11.—Paralysis of the posterior crico-arytenoid. (Inspiration.)

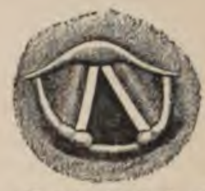

FIG. 12.—Paralysis of the right recurrent laryngeal nerve. (Inspiration.)

Paralysis of the recurrent laryngeal nerve: the affected vocal band is completely immobile in the cadaveric attitude, while the unparalyzed band exhibits an abnormally wide range of movement. The voice is feeble, but there is no dyspnea. When the paralysis is bilateral corresponding conditions are found: aphonia, no dyspnea, toneless cough. Causes: neuritis; compression by an aneurism of the aorta or a goiter; attends tabes, etc.

Paralysis of the epiglottis (postdiphtheric) is attended with difficulty in swallowing, together with cough.

3. Lungs.

Anatomic.

The lungs are contained air-tight within the pleural cavities. The right lung has three (upper, middle, and lower) lobes, the left but two (upper and lower). The pleural cavities are not entirely occupied by the lungs, as the reflection of the serous membrane extends a greater distance (up to 9 cm.—3½ in.) below on both sides, and at the cardiac fissure upon the left, resulting in the formation of the complementary spaces, which are almost filled by the lungs only upon the deepest inspiration. The base of the lung is applied directly to the diaphragm.

The *topographic relations* of the lungs with the thoracic skeleton are of importance. Anteriorly the ribs are used as landmarks for the indication of varying levels, while upon the back the spinous processes of the vertebræ from the seventh cervical downward serve a similar purpose. Other coördinates sometimes employed to indicate the lateral relations of the lungs are furnished by a number of imaginary vertical lines: the median line, through the middle of the sternum; the sternal line, through the sternal margin; the mammillary line, through the nipple; between the last two is the parasternal line; finally there are the anterior, middle, and posterior axillary lines (the last passing through the lower angle of the scapula).

Right Lung.—Upper limit: anteriorly, from 2 to 4 cm. (¾–1½ in.) above the clavicle; posteriorly, at the level of the seventh cervical vertebra.

Median limit: somewhat to the left of the median line to the insertion of the sixth rib.

Lower limit: lower border of the sixth rib (mammillary line), lower border of the seventh rib (anterior axillary line), level of the ninth rib (scapular line), level of the spinous process of the eleventh dorsal vertebra (posteriorly).

The boundary between the right upper and middle lobes, anteriorly, corresponds with the upper border of the fourth rib; posteriorly, it pursues an oblique course from the third dorsal vertebra downward and outward.

The **Left Lung** has corresponding relations, the upper lobe reaching anteriorly to the sixth rib (see Plate 23 *et seq.*). The level of the diaphragm corresponds with the insertion of the fourth rib during deep expiration. It is somewhat higher upon the right side than upon the left. (For further particulars see Plates 23–25.)

During respiration the margins of the lungs move in the mammillary line a distance of from 2 to 4 cm. ($\frac{3}{4}$ to $1\frac{1}{2}$ in.). The respiratory mobility is greatest in the axillary line (10 cm.—4 in.).

Physiologic.

During respiration the lungs engage in no active movement. The motor energy is furnished by the diaphragm and the thoracic muscles, and the lungs follow passively the dilatation of the thorax thus effected.

The respiratory interchange of gases takes place between the alveoli of the lungs and the pulmonary capillaries. The inspired air contains 79 per cent. of nitrogen, 21 per cent. of oxygen, and 0.04 per cent. of carbon dioxid, together with watery vapor taken up in the air-passages (nose, larynx). The expired air contains a larger proportion of carbon dioxid (4 per cent.), less oxygen (16 per cent.), and is saturated with watery vapor. From 900 to 1200 grams of carbon dioxid are expired daily (the larger amounts under conditions of greater activity). When the interchange of gases is in any way deranged in consequence of disease of the air-passages, the alveoli or the capillary circulation (diseases of the heart, diseases of the blood), the deficiency must be made good as far as possible by increased activity of the respiratory processes, and there thus results dyspnea.

Spirometry.

The vital capacity of the lungs—that is, the amount air that can be inhaled on deep inspiration after the de est possible expiration—equals in men from 3000 to 4C cu.cm.; in women from 2000 to 3000 cu.cm. The p monary capacity is measured by means of a spiromet It is diminished pathologically in conjunction with diseases of the lungs and with meteorism.

Inspection.

Before entering upon any other examination obser tion should be directed to the degree of development, form, and the symmetry of the thorax, the character the respiration, etc. (For particulars see p. 13.)

Percussion of the Lungs. (For theoretic conside tions see p. 29.)

Percussion is practised first upon the right, then up the left—

1. In the supraclavicular fossa (preferably from behi forward);
2. In the individual intercostal spaces in the mamm lary line downward;
3. Over the back from above downward in successi areas a hand's breath from the middle line until upon right absolute dulness is reached, the note at each le on the right side being compared with the correspondi note upon the left.

Over the normal lung is yielded *pulmonary resona* —that is, a clear, not tympanitic note, extending upon right to the lower border of the sixth rib. Below t point is the liver-dulness. Upon the left side pulmon resonance extends to about the same level as on the rig although this limit is somewhat difficult of determinati on account of the presence of the stomach, likewise c taining air, though yielding a tympanitic note. Up the back pulmonary resonance continues to the level the spinous process of the eleventh dorsal vertebra.

Displacement upward of the lower limits of the lu (from elevation of the diaphragm) occurs in conjuncti with meteorism, ascites, tumors of the abdomen (bilateral); also with contraction of the lungs and the pleura (unilateral). *Displacement downward* of the limits of the lungs (to the ninth rib) occurs, apart from the respiratory displacement, in conjunction with pulmonary emphysema (permanent) and asthma (transient).

The normal respiratory displacement may be entirely wanting if the lungs are adherent, and in cases of extreme emphysema.

Dulness upon percussion over the lungs may result from:

1. Infiltration of the lungs (filling up of the alveoli with liquid or solid masses, which displace the air), which occurs in conjunction with pneumonia, tuberculosis, infarction, abscess-formation, gangrene, tumors;
2. Atelectasis, absence of air in consequence of compression of the lungs, or as a result of spontaneous absorption of the air, with insufficient supply (occluded bronchi);
3. Displacement of the lungs from the walls of the thorax, in consequence of accumulation of fluid in the pleural cavity (pleuritic exudate, hydrothorax), and of the presence of dense adhesions and of tumors.

Together with the dulness associated with the conditions named in the third group, the percussing finger appreciates a sense of marked resistance.

An infiltrated area of lung must have an extent of at least 4 cm., and an accumulation of fluid must equal at least 400 cu.cm., in order to be distinctly demonstrable by dulness on percussion.

Dulness over the apices is suggestive rather of tuberculosis; over the lower lobes, of pneumonia or pleurisy.

The upper limit of pleuritic dulness pursues an oblique course from above and behind, forward and downward, in consequence of the posture of the body at the time when the exudation took place.

When air and fluid are simultaneously present in the pleural cavity (sero-pyopneumothorax) the level and the plane of the fluid vary with the posture of the patient. This change in level is wanting in connection with pleuritic effusions, in consequence of inflammatory adhesions.

Symptoms of Displacement of the Lungs.—When fluid and air gain entrance into the pleural cavity, as well as when tumors are present, adjacent organs are displaced from their normal situations. Upon the right side the liver is pushed downward, the heart and the mediastinum toward the left, while upon the left side the heart is displaced toward the right. The complementary pleural spaces become filled with fluid. These evidences of displacement are readily demonstrable by percussion, the normal limits of dulness of heart and liver being displaced. Although the dulness due to a pleural effusion upon the right side is not to be separated from that of the liver, similar dulness upon the left side can be readily separated from the tympanitic note of the contiguous stomach. This tympanitic area normally present below the left lung (Traube's semilunar space) becomes dull upon percussion in its upper half when left-sided exudative pleurisy exists. This physical sign aids in the differentiation of the last-named condition from left-sided pneumonia, in conjunction with which the complementary space naturally remains uninvolved, and Traube's space retains the normal tympanitic note of the stomach.

A tympanitic note over the lungs is found—

1. Above the level of pleuritic effusions, from loss of pulmonary tension;
2. Over pneumonic and tuberculous infiltrates (dull-tympanitic);
3. When abnormal excavations have formed (caverns), and have attained at least the size of a walnut (tuberculosis, bronchiectasis, gangrene);
4. At times in cases of pneumothorax, if the tension of the contained air be not too great. When the tension is high, as it is the more commonly, the note is abnormally full, deep, and not tympanitic.

A distinction is to be made between the high tympanitic and the deep tympanitic note, in accordance with the size of the cavity.

A metallic note occurs—

1. Over very large cavities with smooth walls (as large as a fist);

2. In cases of pneumothorax (pleximeter-rod percussion, see p. 30).

The *cracked-pot sound* occurs when upon deep percussion the air is compelled to escape through a narrow opening (stenotic murmur). It is found:

1. Over caverns communicating by a small opening with the bronchi;

2. Over relaxed or infiltrated pulmonary tissue (rare).

Normally it is heard frequently upon percussion of crying children.

Ringing sounds sometimes resemble the tinkling of coins.

Variations in the percussion-note.

By these are understood changes from a tympanitic note to one of higher or lower pitch.

1. Wintrich's change in percussion-note. On opening the mouth a higher note, on closing the mouth a deeper note is developed. This is heard over caverns and over pneumothorax when direct communication with a bronchus exists. It is present normally upon percussion over the larynx.

2. Friedreich's respiratory change in percussion-note. Over caverns the note under some circumstances becomes higher on deep inspiration.

3. Gerhardt's postural change in percussion-note. When caverns are filled partly with air and partly with fluid the note becomes deeper or higher on change of position (sitting, recumbency), in accordance with the direction of the longest diameter of the cavity.

4. Biermer's change in percussion-note. In cases of sero-pneumothorax the note becomes deeper when the patient sits up, in consequence of enlargement of the air-space.

Auscultation of the Lungs.

This is practised in the same situations and in the same manner as percussion of the lungs, comparing the sides with one another.

At the apices of the lungs anteriorly (supraclavicular fossæ) auscultation may be more advantageously practised with the aid of a stethoscope. In the remaining portions direct auscultation is equally applicable.

Over the healthy lung is heard everywhere pure vesicular breathing (vesicular inspiration, short expiration). Only at the upper portion of the right side posteriorly is the blowing breathing of trachea and bronchus to be heard.

In children the breathing is particularly loud and rough (puerile breathing).

In the region of the heart a slight exaggeration of the breathing takes place with every systole (systolic vesicular breathing).

Pathologic Vesicular Breathing.—*Exaggerated vesicular breathing* is found when resistance is encountered in the bronchi, as from swelling or accumulation of secretion, and in cases of bronchitis.

Enfeebled vesicular breathing occurs in conjunction with occlusion of the bronchi (catarrh), with pulmonary emphysema (diminished aëration of the lungs), over small pleuritic effusions (displacement of the lung from the chest-wall).

Prolongation and accentuation of expiration take place when resistance is present in the bronchi (swelling, narrowing, accumulation of mucus), in cases of bronchitis, of asthma, and of pulmonary emphysema.

Jerking inspiration is especially common over the apices of the lungs in cases of beginning pulmonary tuberculosis.

Bronchial breathing is heard—

1. When infiltration of the lung exists (pneumonia, tuberculosis, gangrene, actinomycosis);
2. When the lungs are compressed (by large effusions),

if, as a result, vesicular breathing is abolished and only the tubular breathing of the bronchi is audible;

3. Over cavities communicating with a bronchus.

Over the apex of the lung bronchial breathing usually signifies tuberculous involvement; over the lower lobe, frequently pneumonia, less commonly bronchiectasis, pleurisy, gangrene.

Amphoric breathing is a ringing, blowing breathing that occurs over large cavities with smooth walls and over open pneumothorax (together with metallic râles and a metallic note upon percussion).

Metamorphosing breathing (over caverns) begins as vesicular breathing, and then becomes bronchial.

Undefined breathing (neither vesicular nor bronchial) occurs when aëration is imperfect at the apices of the lungs under both normal and pathologic conditions (beginning tuberculosis, pleuritic adhesions).

Absence of breath-sounds is noted over large pleuritic exudates, over closed pneumothorax, and temporarily when the bronchi are occluded.

Râles occur in greater or lesser number when mucus, blood, or pus is present in bronchi and in caverns.

Dry râles (coarse, snoring, whistling râles) occur when the secretion is viscid. Whistling is indicative of catarrh or stenosis of the smallest bronchi.

Moist râles (small, medium-sized, large) occur when the secretion is thin and liquid, varying in accordance with the size of the bronchi. Large moist râles are indicative of caverns.

Crepitant râles are generated by the sudden separation of the walls of alveoli and bronchioles previously glued by secretion, and occur in conjunction with atelectasis, beginning pneumonic infiltration, and beginning resolution (crepitatio indux and crepitatio redux), in cases of pulmonary edema, of miliary tuberculosis, and of compression of the lung. Not uncommonly they attend the first deep inspirations, even upon auscultation of healthy lungs, especially low down posteriorly.

Ringing râles are heard over caverns and extensive compression and infiltration of the lungs (transmission from the large bronchi).

Metallic râles are described under amphoric breathing (p. 47).

Hippocratic succussion (metallic splashing-sounds) may be heard at some distance from the patient in cases of sero-pneumothorax and of pyo-pneumothorax upon vigorous shaking. Under these conditions there develops also *metallic tinkling.*

Pleuritic friction-sounds occur most frequently in association with dry, fibrinous pleurisy, disappearing when exudation takes place and recurring with absorption of the fluid. They are usually heard loudly over the lower lobes, especially upon their lateral aspects. They are naturally absent in case of adhesive pleurisy. Friction-sounds may be present when deposition of tubercles takes place upon the pleura. The sounds become louder upon deep inspiration.

Auscultation of the Voice, Pectoral or Vocal Fremitus.—*Enfeeblement* of the transmitted voice (upon counting 99, etc.) for both ear and hand occurs—

1. When the bronchi through which the sound is transmitted are occluded (by secretion, tumors, or stenosis);
2. When either fluid or air has found its way between the lungs and the chest-wall (pleurisy, hydrothorax, pneumothorax);
3. When the voice is enfeebled.

Exaggeration of the transmitted voice for the ear (bronchophony; when marked, pectoriloquy; when attended with a bleating sound, egophony) and for the hand (increased pectoral fremitus, marked vibration) occurs in association with condensation of the pulmonary tissue (pneumonia, above pleuritic exudates, over caverns with dense walls, also over pleuritic thickening).

4. Puncture of the Pleura.

When symptoms are present indicative of a pleural effusion: boardlike dulness, especially over the lower portions of the chest, with a sense of resistance; enfeebled breathing, with crepitant râles; abolished pectoral fremitus, with a tympanitic note above the dulness; displacement of liver and heart (if right-sided); displacement of heart, obliteration of Traube's semilunar space (if left-sided)—resort may be had to exploratory puncture to determine the character of the effusion, especially if the dulness continues to increase.

The needle is usually introduced in the seventh, eighth, or ninth intercostal space one or two hands'-breadth from the vertebral column (for technic, see page 24). The fluid thus obtained may be—

1. Serous, light yellowish (pneumonia, sepsis, rheumatic pleurisy, tuberculous pleurisy);
2. Hemorrhagic (carcinomatous pleurisy, tuberculous pleurisy);
3. Purulent (empyema);
4. Putrid (gangrene, from intestinal perforation).

Microscopic examination (carcinoma-cells), and especially bacteriologic examination (see p. 24), including staining, culture, and finally inoculation (tubercle-bacilli), are further of importance. There may be found staphylococci, streptococci, diplococci, bacterium coli, tubercle-bacilli,[1] actinomyces-fungus.

If the pleurisy have terminated, the introduced needle encounters only dense thickening and adhesions. The development of this condition is recognized by the contraction and retraction of the corresponding portion of the chest. A sacculated exudate may, however, persist for a long time in the midst of such adhesions.

Exudates are rich in albumin (coagulation on heating) and fibrin (spontaneous coagulation).

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[1] Negative bacteriologic findings do not exclude tuberculous pleurisy, the exudate attending this condition being usually sterile.

Transudates, on the other hand, do not coagulate, or at most become slightly turbid when heated.

The specific gravity of exudates is usually above 1018, that of transudates below 1015.

5. The Sputum.

The presence of fluid in the bronchi induces reflex cough through irritation of the sensory bronchial nerves (vagus), with expulsion of the irritating material.

Cough is a complicated expiratory movement. The chink of the glottis is first closed spasmodically, so that the bronchial column of air is exposed to a higher degree of pressure through the increased expiratory movement. Then sudden opening of the glottis permits explosive escape of the air, which carries any sputum present with it.

Cough is rendered difficult or impossible when the respiratory muscles or the adductors of the vocal bands are paralyzed; when severe pain is present (pleurisy, pneumonia); when marked weakness exists; and in states of coma (development of catarrhal pneumonia or of pulmonary edema).

The *expectoration* is constituted of—

1. The secretion of the alveoli and of the bronchial and laryngeal mucous membranes;
2. Certain inspired elements (dust, carbon);
3. Material derived from cavities and perforations (pus, putrid matters, blood, portions of food);
4. Admixture from pharynx, mouth, and nares.

The following **varieties of sputum** are to be distinguished:

1. Pure mucous sputum (sticky, glassy), observed in cases of bronchitis and of beginning tuberculosis;
2. Pure purulent sputum (thickish, confluent, yellowish), observed in cases of broncho-blennorrhea, of abscess of the lung, of rupture of empyemata, etc.;
3. Mucopurulent sputum (globular, nummular), observed in cases of bronchitis, of broncho-blennorrhea, and of tuberculosis;

4. Serous sputum (quite diffluent and frothy), observed in cases of pulmonary edema (with slight admixture of blood : prune-juice like) ;

5. Hemorrhagic sputum, observed—

a. In cases of croupous pneumonia (bloody-mucous, rust-brown, tough, viscid) ;

b. In cases of pulmonary infarction (hemorrhagic infarction from pulmonary embolism) ;

c. In association with marked stasis in the pulmonary circulation (cardiac lesions) ;

d. In conjunction with violent paroxysms of cough, derived from pharynx or larynx ;

e. In the form of pure blood, bright-red, frothy sputum (hemoptysis), from erosion of blood-vessels through ulcerative processes, in cases of pulmonary tuberculosis, of bronchiectasis, and of gangrene, and less commonly in cases of malignant tumors, of rupture of an aortic aneurism, and of rupture of varices in the bronchi ;

f. In cases of simulation and of hysteria (bleeding from the gums).

Fibrinous coagula (bronchial casts) occur in association with pneumonia, diphtheria, and fibrinous bronchitis.

Sputum in layers.

When sputum that is partly serous, partly mucous, partly purulent, and abundant (bronchorrhea, bronchiectasis), is placed in a beaker it separates into distinct layers : an upper frothy mucous, a middle serous, and a lower purulent layer.

Color.—The sputum may be—

Red, from admixture of blood ;

Brown, from disintegration of blood present ;

Ocher-yellow, in cases of perforating abscess of the liver (bilirubin) and of abscess of the lung (hematoidin) ;

Egg-yellow, from the action of bacteria (sarcinæ) in purulent sputum ;

Blackish, from admixture of carbon-particles ;

Greenish, in cases of pneumonia with delayed resolution and in cases of bilious pneumonia (icterus).

A *putrid* odor is present in cases of fetid bronchitis and of gangrene, from the presence of putrefactive bacteria.

Microscopy (see Plates 7 to 10. Technic, see p. 21).

Sputum contains *normally*—

a. Epithelial cells:

Squamous epithelium from the mouth and from the true vocal bands;

Cylindric epithelium (uncommon) from the nose, the larynx, the trachea, and the bronchi;

Alveolar epithelium (large round cells filled with myelin, fat-drops, and carbon-particles), of doubtful origin;

b. White blood-corpuscles (in a state of more or less advanced fatty degeneration) from all portions of the respiratory tract;

c. Red blood-corpuscles (quite isolated);

d. Mucin-threads (nose, bronchi);

e. Bacteria (cocci, sarcinæ, bacilli, mycelial fungi from the mouth, molds from the nose).

Pathologically, there may be present—

a. Cellular and tissue elements:

Leukocytes in all stages of disintegration, containing fat and coloring-matter (rupture of abscess);

Eosinophile cells are numerous in cases of asthma, and isolated in cases of chronic bronchitis. These are large cells filled with bright, yellowish granules (*α*-granules), which become distinctly visible when stained with eosin. They may be stained with methylene-blue and eosin, and become especially distinct when briefly immersed in 10 per cent. potassic hydroxid after being stained.

Cardiac-lesion cells, in connection with chronic pulmonary stasis (especially mitral stenosis). These are large cells filled with yellowish-brown pigment-granules derived from hemoglobin (alveolar epithelial cells and leukocytes ?).

Shreds of lung-tissue, discolored particles consisting of detritus, lung-black, leukocytes, bacteria, fat-crystals, fat-drops, occurring in cases of pulmonary gangrene and of pulmonary abscess.

Elastic fibers (bright-yellow filaments of double contour) are found in connection with all destructive processes in the lungs (tuberculosis, pulmonary abscess); they are usually absent in cases of pulmonary gangrene, in consequence of the solvent action of a ferment present. They become especially distinct upon addition of 10 per cent. potassic hydroxid, and are usually found in the caseous plugs (lentils) of tuberculosis.

Curschmann's spirals (corkscrew-like filaments of mucus wound about a central thread) are formed in the bronchioles in cases of bronchial asthma, of capillary bronchitis (through exudation ?). They are present generally in the sago-masses, and are most readily distinguished when the sputum is spread in a thin layer upon a black background.

Echinococcus-hooklets and vesicles occur in cases of echinococcus of the lung or of rupture of an echinococcus-cyst of liver, kidney, etc.

b. Crystals:

Needles and spheres of fatty acids (gangrene, fetid bronchitis, abscess) are present in the so-called Dittrich's plugs (whitish-yellow, friable, fetid granules).

Charcot-Leyden crystals, pointed vitreous octahedra (accidentally in cases of bronchial asthma).

Cholesterin-plates, leucin-spheres, tyrosin-needles, in cases of pulmonary abscess, of fetid bronchitis, and of bronchiectasis.

Hematoidin-crystals (brownish-red rhombic plates, spheres, needles), following hemorrhages in cases of abscess of the lung or rupture of an abscess from a neighboring organ. They may be free or enclosed within cells.

c. Bacteria (for staining technic, see p. 24).

Tubercle-bacilli (for demonstration, see p. 25). Even when present in small numbers only, the diagnosis is established. Negative findings are not conclusive. Tubercle-bacilli are to be found with greatest certainty in the so-called lentils of the sputum.

Influenza-bacilli.—Stain with magenta-red. They are usually demonstrable in large numbers in cases of influenza-catarrh and in influenza-pneumonia.

Pneumococci.—Stain by Gram's method. They are found abundantly in the sputum in cases of pneumonia, but occur also in the sputum of healthy persons.

Actinomyces-granules. — These are distinctly visible macroscopically as yellow granules when viewed upon a black background.

Anthrax-bacilli have been found in the sputum of the infrequent cases of pulmonary anthrax.

Aspergillus Mycelium (molds) is found in cases of aspergillous pneumonomycosis (secondary infection in old cases of tuberculosis, pulmonary infection, etc.).

Thrush-fungus (oïdium) is found in the mouth, esophagus, etc., of children and debilitated individuals.

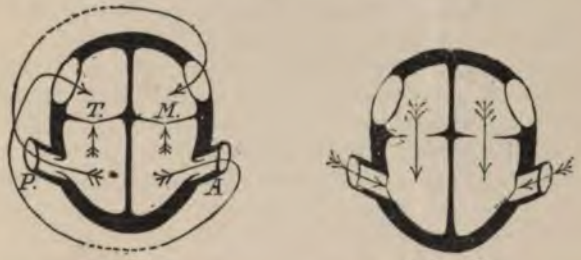
For details concerning these parasites, see Section VI.

II. EXAMINATION OF THE CIRCULATORY APPARATUS.

1. Heart.

Anatomic.—The heart is a hollow muscular organ, lying obliquely upon the diaphragm and contained within the pericardium. It is contained two-thirds in the left and one-third in the right half of the body, and extends from the lower border of the second rib at its insertion into the sternum to the upper border of the sixth costal cartilage. It is in large part covered by the median margins of the lungs, and is in direct contact with the chest-wall throughout only a small extent (to the left of the sternum, from the fourth to the sixth intercostal space, cardiac fissure of the left lung). Above and to the right is situated the base of the heart, with the related large vessels, while the apex is situated below and to the left in the fifth intercostal space, somewhat internal to the left mammillary line.

Physiologic.—The activity of the heart furnishes the driving-force for the circulation of the blood; the accompanying diagrams (Fig. 13) illustrate this mechanism. Fig. 13, *a*, shows the position of the valves of the heart during the contraction of the ventricles (systole); Fig. 13, *b*, their position during relaxation (diastole).



a, systole. *b*, diastole.

FIG. 13.—Diagrammatic representation of the normal action of the heart (showing position of the valves, direction of the blood-stream, and degree of blood-pressure).

During the ventricular *systole* the aortic and pulmonary valves are open, while the mitral and tricuspid are closed. The blood passes out of the left ventricle through the aorta into the arteries, capillaries, and veins of the body to the right auricle; also from the right ventricle, through the pulmonary artery, into the pulmonary arteries, capillaries, and veins to the left auricle. During the ventricular *diastole* the mitral and tricuspid valves are open, while the aortic and pulmonary are closed. The blood courses from the left auricle into the left ventricle, from the right auricle into the right ventricle. The direction of the prevailing blood-pressure is indicated by the arrows. It is thus obvious that without exact functional activity of the valvular mechanism circulatory disturbances must at once arise.

Theory of Valvular Lesions.—If a heart-valve does not close completely at the proper time (*insufficiency of the valve*), a current of blood will flow abnormally through the unclosed orifice. Absolute insufficiency results from contraction of the free extremity of a valveleaflet, relative insufficiency from abnormal dilatation of an orifice, so that the normal valve is no longer sufficient to effect its perfect closure.

When an orifice of the heart is narrowed pathologically (*stenosis of the orifice*) a smaller amount of blood will pass in a unit of time. Stenosis occurs from circular calcification, adhesion, and contraction of valves and orifices.

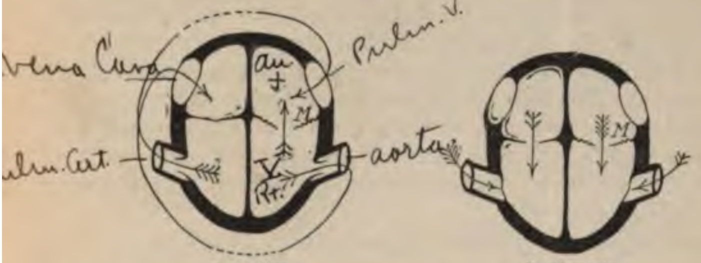
As a result of the conditions named, one portion of the heart will either receive too much blood from an abnormal source (insufficiency), or, if it cannot adequately expel its contents (stenosis), it will retain too much blood. In either event there results blood-stasis, and in consequence dilatation of the respective heart-cavity. If this be not neutralized by increased activity of that portion of the heart, it may lead to further stasis in the circulation physiologically behind it. Such neutralization is effected through *hypertrophy* of the muscular wall of the affected segment. If the further injurious action of the derangement be thus overcome, the resulting condition is designated *compensation* of the cardiac lesion. The establishment of compensation implies that the blood-pressure relations in the general and pulmonary circulation are restored to the normal.

A distinction is to be made between primary and secondary hypertrophy. If a cavity of the heart be dilated, the effects may be neutralized by secondary hypertrophy. Primary hypertrophy, on the other hand, develops in response to simple increase in the pressure to be overcome, without simultaneous dilatation.

The most important valvular lesions are—

1. *Insufficiency of the mitral valve* (Fig. 14).

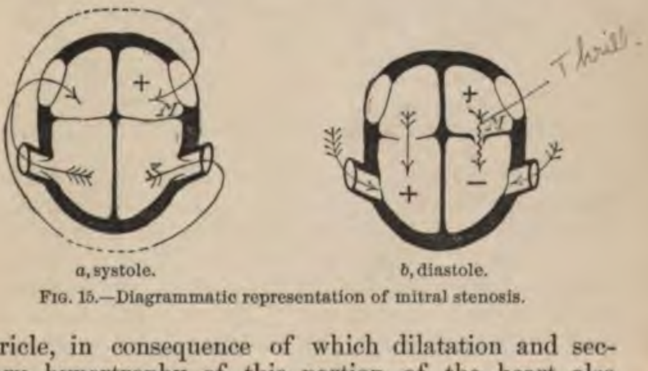
During the ventricular systole (Fig. 14, *a*) the blood passes from the left ventricle not only into the aorta, but, in consequence of insufficiency of the mitral valve, also into the left auricle, with the generation of a systolic murmur. The left auricle thus receives blood from two sources during diastole, and in consequence dilatation and secondary hypertrophy result. The stasis extends to the pulmonary circulation, with increase in the blood-pressure; as a result a greater amount of work is required of the right ventricle, which undergoes primary hypertrophy.



a, systole. *b*, diastole.

FIG. 14.—Diagrammatic representation of mitral insufficiency (pathologic increase of pressure is indicated by arrows). + : + bld.

During the ventricular diastole (Fig. 14, *b*) a larger amount of blood than normal streams out of the dilated and secondarily hypertrophied left auricle into the left ventricle, in consequence of which dilatation and secondary hypertrophy of this portion of the heart also take place.



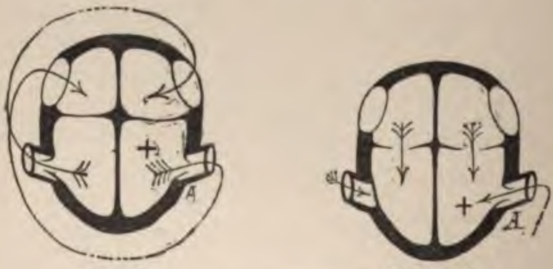
a, systole. *b*, diastole.

FIG. 15.—Diagrammatic representation of mitral stenosis.

2. *Stenosis of the mitral orifice* (Fig. 15).

During the ventricular diastole (Fig. 15, *b*) the blood, in consequence of stenosis at the mitral orifice, passes only with difficulty from the left auricle into the left ventricle, with the development of a ~~diastolic~~ murmur. The left auricle does not empty itself completely, so that dilatation results, with secondary hypertrophy of this part of the heart, and at the same time stasis in the pulmonary circulation, as the left auricle can undergo only a slight degree of hypertrophy, which is insufficient to meet the demands of its increased work. As a result an increased burden is thrown upon the right ventricle during systole, and this chamber undergoes hypertrophy. The left ventricle remains ~~unchanged~~, as it receives a deficiency of blood.

3. *Insufficiency of the aortic valve* (Fig. 16).



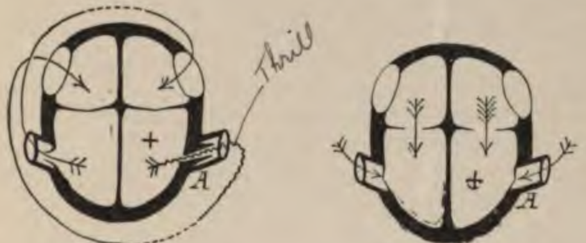
a, systole. *b*, diastole.

FIG. 16.—Diagrammatic representation of aortic insufficiency.

During the ventricular diastole (Fig. 16, *b*) the blood, in consequence of insufficiency of the aortic valve, flows from the aorta back into the left ventricle, with the generation of a diastolic murmur. The ventricle thus receives during the diastole blood from two sources, and in consequence undergoes dilatation. This leads to secondary hypertrophy, which in turn results in more blood being sent into the aorta than before, in order that it may suffer without detriment the loss of blood due to regurgitation during diastole. All other conditions remain unchanged.

4. *Stenosis of the aortic orifice* (Fig. 17).

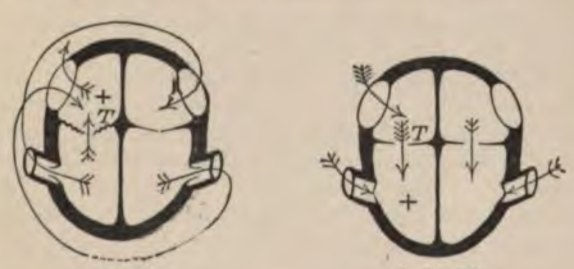
During the ventricular systole (Fig. 17, *a*) the blood, in consequence of stenosis at the aortic orifice, passes only with difficulty into the aorta, with the generation of a ~~systolic murmur~~. Some blood would thus be retained in the left ventricle, so that dilatation would result if the obstruction were not overcome through ~~primary hypertrophy~~ of the left ventricle.



a, systole. *b*, diastole.

FIG. 17.—Diagrammatic representation of aortic stenosis.

5. *Insufficiency of the tricuspid valve* (Fig. 18).



a, systole. *b*, diastole.

FIG. 18.—Diagrammatic representation of tricuspid insufficiency.

During the ventricular systole (Fig. 18, *a*) the blood, in consequence of insufficiency of the tricuspid valve, passes from the right ventricle not only into the pulmonary artery, but also into the right auricle, with the generation of a systolic murmur. The auricle thus receives during its diastole blood from two sources, and undergoes dilatation and secondary hypertrophy.

During the ventricular diastole (Fig. 18, *b*) the right ventricle receives thus more blood than usual. It also undergoes dilatation and secondary hypertrophy, in consequence of which compensation becomes established. As, however, the right auricle, in consequence of its small muscular reserve, soon gives way, the stasis gradually extends also to the venæ cavæ and the veins beyond. With increasing dilatation of the veins their valves become relatively insufficient, and the blood regurgitated into the auricle during the ventricular systole is forced back also into the superior and inferior venæ cavæ, with the generation of a jugular and an hepatic venous pulse. In this way there will be brought about overfilling of the general venous circulation.

The mechanism of the remaining uncommon valvular lesions may be comprehended from the principles laid down.

Derangement of Compensation.

Just as in connection with the valvular lesion last described, so also will there, in conjunction with the other lesions, after compensation has been maintained for some time, occur finally failure of the auricle or of the ventricle, when the muscular power of the hypertrophied heart is no longer sufficient. There then develops dilatation of this segment of the organ, and the result of the irremediable stasis that follows is an overfilling of the venous circulation at the expense of the arterial. As, further, too little blood is arterialized in the lungs, in consequence of the retarded circulation, the blood is deprived of oxygen and does not sufficiently get rid of carbon dioxid, and the result is increased venosity. In turn there is developed dyspnea, which occasions increased respiratory effort as a sort of further compensation, and cyanosis when this form of pulmonary compensation no longer suffices.

In consequence of the venous stasis the blood is not sufficiently emptied out of the capillaries and lymph-channels, and an accumulation of blood-serum, in the form of a transudation through the walls of the vessels, takes place into the interstices of the tissues, with the development of swelling and anasarca. These changes are noted first only in the dependent portions, from the influence of gravitation; then also in the cavities of the body and over the whole body. The venous stasis manifests itself further in swelling of the liver and of the spleen. At the same time, in a vicious circle, the secretion of urine is diminished in consequence of the retarded circulation of blood in the kidney (cyanotic kidney). All of these manifestations together constitute the characteristic picture of derangement of compensation, which may develop, further, in connection with all other diseases of the heart besides valvular lesions.

Inspection and Palpation of the Precordium.

Prominence of the precordium (bulging) is found in conjunction with some valvular lesions, with mitral stenosis, etc. (especially in children), and with exudative pericarditis.

Visible pulsation:

Normal: in the situation of the apex-beat; feeble pulsation in the epigastrium;

Pathologic: in conjunction with contraction of the lung in the second, third, and fourth intercostal spaces, with aneurismal formation; marked epigastric pulsation (dilatation of the right ventricle).

Systolic retraction at the apex of the heart may be present in conjunction with adhesions of the pericardium.

The apex-beat is indicative of the furthest limit of visible and palpable movement of the heart to the left and downward. Its determination furnishes, therefore, an important index: the left border of the heart. Normally it lies in the fifth intercostal space somewhat within the mammillary line. In children it is somewhat higher, in old persons somewhat lower.

Permanent *displacement* of the apex-beat takes place:

1. *Upward*, with elevation of the diaphragm (meteorism, ascites, abdominal tumors, gravidity);
2. *Downward*, with hypertrophy of the left ventricle, with aortic aneurism, with depression of the diaphragm (pleurisy, pneumothorax);
3. Toward the *left*, with dilatation and hypertrophy of the left ventricle, with displacement (right-sided exudate or pneumothorax), with left-sided pleural contraction (traction);
4. Toward the *right*, with displacement (left-sided exudate or pneumothorax), or through right-sided contractions.

Temporary displacements, particularly toward the left, occur in the lateral decubitus.

Enfeeblement of the apex-beat occurs in conjunction with obesity, overlying of the lungs, feebleness of the heart, pericarditic effusion (the apex-beat lying within the left limit of dulness).

Augmentation of the apex-beat occurs in connection with unusual physical effort, febrile states, hypertrophy and dilatation of the heart.

Thrill in the precordium may be felt in connection with:

a. Stenotic murmurs (purring tremor): with mitral stenosis, diastolic; with aortic stenosis, systolic;

b. Aneurism of the aorta or of the pulmonary artery;

c. Pericarditic friction.

Percussion of the Heart.—Percussion (light) is practised to determine—

The *upper limit* of cardiac dulness. The examination is begun in the left sternal line, with the finger applied horizontally, parallel with the intercostal spaces, and passing from above downward. Normally, this limit is found at the lower border of the fourth rib.

The *right limit* of cardiac dulness. The finger is placed vertically external to the right mammillary line at the level of the fifth or sixth rib and passing from right to left. Normally, this limit is found at the left margin of the sternum.

The *left limit* of cardiac dulness is studied in the same way as the right, except that the finger is placed first in the axillary line, passing toward the right. Normally, it is found somewhat internal to the left mammillary line (in the same situation as the apex-beat).

In this manner, passing from the area yielding a ~~clear~~ note to that yielding a ~~dull note~~, the *absolute cardiac dulness* is determined; that is, that portion of the chest to which the heart is directly applied without interposition of pulmonary tissue. (For details see Plate 23.)

The absolute cardiac dulness is surrounded by a finger's-breadth zone of *relative dulness;* that is, that portion of the chest which upon deep percussion yields uncertain dulness, by reason of the interposition of the margins of the lungs between the heart and the chest-wall. This has but little significance.

Inferiorly, the cardiac dulness passes without differentiation into that of the liver.

Increase in the area of absolute ~~cardiac dulness~~ occurs—

1. Toward the *left*, in conjunction with hypertrophy and dilatation of the left ventricle, with displacement (exudation), with left-sided retraction of the lung;

2. Toward the *right*, in conjunction with dilatation of the right ventricle, with accumulation of fat beneath the sternum, with displacement;

3. Toward the *left and right,* in conjunction with exudative pericarditis, hydropericardium (triangular area of dulness, with the apex at the manubrium sterni), contraction of the lung, elevation of the diaphragm.

Diminution in the area of cardiac dulness occurs in connection with pulmonary emphysema (becoming more distinct when the body is bent forward) in consequence of overlying by the distended lungs.

In cases of pneumopericardium the cardiac dulness is replaced by a tympanitic or metallic note.

Abnormal dulness attends—

1. Patulousness of the duct of Botal: a small quadrilateral area of dulness superimposed upon the cardiac dulness;

2. Aneurism of the aorta: an area of dulness at the level of insertion of the second and third ribs upon the right, either separated from or continuous with the cardiac dulness.

3. Mediastinal tumors: quite irregular areas of dulness over the sternum.

Auscultation of the Heart (for theoretic considerations see p. 33).

The four points of auscultation (points of greatest intensity of the heart-sounds) are—

For the *mitral* sounds: the apex of the heart.

For the *tricuspid* sounds: the insertion of the fifth right rib.

For the *aortic* sounds: the second intercostal space to the right of the sternal margin.

For the *pulmonary* sounds: the second intercostal space to the left of the sternal margin.

In each of these situations are heard two sounds, a systolic and a diastolic. At the venous orifices the systolic (**duk** duk), at the arterial orifices the diastolic sound (duk **duk**) is accentuated.

Abnormal accentuation of the first mitral sound occurs in conjunction with increased activity of the heart in febrile states, with chlorosis, neurasthenia, hypertrophy of the left ventricle.

Abnormal accentuation of the second aortic sound occurs in conjunction with hypertrophy of the left ventricle (nephritis, cardiac lesion).

Abnormal accentuation of the second pulmonary sound occurs in conjunction with hypertrophy of the right ventricle, and is an important sign of increase of blood-pressure in the pulmonary circulation.

In consequence of relaxation of the right ventricle the accentuation of the second pulmonary sound is less distinct with all derangements of compensation than with efficient compensation. The remaining sounds also may, under these conditions, be fainter and less distinct.

Metallic heart-sounds occur in conjunction with pneumopericardium and with caverns in the lung.

Division (*duplication*) of the first sound at the apex occurs in connection with hypertrophy of the heart (chronic nephritis); a presystolic sound (auricular contraction), with mitral stenosis (although a murmur may be wanting); division of the second sound over the large vessels (asynchronous valvular closure, with disturbance in the coincidence of ventricular activity) occurs under variable conditions (fever, disease of the heart, and also even in health).

Galloping rhythm (triple heart-sounds: duk **duk** duk) occurs in conjunction with weakness of the heart in consequence of asynchronous ventricular activity, and is a sign of ill omen.

Heart-murmurs (for theoretic consideration see p. 33).

Systolic murmurs occur between the beginning of the first and that of the second sound.

Diastolic murmurs occur between the beginning of the second and that of the first sound. Diastolic murmurs heard shortly before the systolic sound are designated *presystolic.*

The heart-sounds may be heard simultaneously with or in advance of the murmur, or not at all. The intensity of the murmur is not a certain guide as to the gravity of the valvular lesion. Diastolic murmurs are usually faint, but of greater significance than possible coincident systolic murmurs.

Murmurs are referred to the valves or orifices in the area of whose sounds (points of auscultation) they are best heard, with the exception of the murmur due to aortic insufficiency.

Systolic (blowing) *murmurs* at the mitral and tricuspid orifices occur in conjunction with insufficiency; at the aorta and pulmonary artery, with stenosis. (See the diagrammatic representations of heart-lesions, p. 55 *et seq.*)

Diastolic murmurs at the mitral (frequently also presystolic and rumbling) and tricuspid orifices occur as a result of stenosis; at the aorta and pulmonary artery, as a result of insufficiency. Aortic diastolic murmurs (rushing) are heard best to the left of the sternum at the attachment of the third rib, thus somewhat lower than the normal area of auscultation, in accordance with the direction of the regurgitant blood-stream.

Accidental systolic murmurs (often loud and blowing) are heard especially at the base of the heart, and are unattended with any other symptom of a valvular lesion, such as dilatation, hypertrophy, or alteration of pulse.

Pericardial murmurs are heard best over the upper portion of the sternum. They do not confine themselves to systole or diastole, but follow intermittently, are scratching, often quadruple (locomotive murmur), seeming nearer to the ear than endocardial murmurs, and losing in distinctness on deep inspiration.

Extra-pericardial (pleuritic) murmurs resemble pleuritic friction (creaking), disappearing generally when the breath is held after deep inspiration or deep expiration. Crepitation synchronous with the action of the heart is heard in conjunction with emphysema of the mediastinum.

2. The Blood-vessels.

Auscultation of the carotid artery is practised at the middle of the inner border of the sterno-cleido-mastoid muscle, and of the subclavian artery in the supraclavicular fossa (lateral segment).

Normally, there are heard over these large vessels a cardiac systolic sound, due to tension of their walls, and a cardiac diastolic sound, due to transmission of the aortic second sound. The cardiac systole corresponds with the arterial diastole, and *vice versâ.*

Systolic murmurs occur in the carotid in conjunction with aortic insufficiency, from entrance of the blood under high pressure; with aortic stenosis and arteriosclerosis, and in febrile states.

Over the small arteries, normally free from sounds if heavy pressure be not made with the stethoscope, a distinct sound is sometimes heard in connection with aortic insufficiency (palmar arch, ulnar artery).

A double sound over the femoral artery is sometimes heard in association with aortic insufficiency, mitral stenosis, lead-poisoning, in consequence of increased arterial pressure.

Duroziez's double sound is heard over the femoral artery (in conjunction with aortic insufficiency) when a certain degree of pressure is made with the stethoscope. It appears as a loud systolic and a faint diastolic blow.

Murmurs over the vessels are heard also in conjunction with aneurismal formations.

The Veins.

The **jugular veins**, at the outer margin of the sterno-cleido-mastoid muscles, exhibit pulsatile increase and diminution in size (diastolic, *presystolic venous pulse*) in conjunction with lesions of the heart.

The *true systolic venous* pulse is observed in connection with tricuspid insufficiency. (See p. 60.) At the same time there exists also visible pulsation of the liver (systolic hepatic venous pulse).

Upon auscultation of the jugular vein in chlorotic patients a *venous hum* may be heard, which is increased on torsion of the head. It may in rare cases be heard also over the femoral vein.

The Pulse.

Frequency.—Normally, it ranges between 60 and 80; in children, over 100, up to 140.

Acceleration (tachycardia, pulsus frequens) occurs in connection with excessive muscular exertion, with excitement, following meals, in febrile states, in connection with diseases of the heart (particularly with derangements of compensation), neurasthenia (in paroxysms), exophthalmic goiter, in states of collapse (over 160), and with paralysis of the vagus.

Retardation (bradycardia, pulsus rarus) attends irritation of the vagus (meningitis, cerebral compression), diseases of the myocardium, aortic stenosis; and occurs as a result of the action of digitalis.

Rhythm.—*Irregular pulse* (intermittent pulse) attends cardiac lesions (especially derangements of compensation and mitral stenosis), sclerosis of the coronary arteries, adhesive pericarditis, etc.

Alternating pulse (one pulse-beat to every two contractions of the heart).

Bigeminate, trigeminate pulse (omission of every third or every fourth beat respectively).

Paradoxic pulse (becoming smaller during inspiration) accompanies pericardial adhesions.

Inequality in size of the radial pulses is noticed in conjunction with aneurisms, embolism of the brachial artery, inequality in caliber of the artery.

Celerity (rapid or tardy ascent of the pulse-wave): quick pulse (pulsus celer), slow pulse (pulsus tardus).

Pulsus celer (running) attends aortic insufficiency, lead-poisoning, interstitial nephritis (increased arterial pressure).

Pulsus tardus attends aortic and mitral stenosis, arteriosclerosis, aneurism.

Size (pulsus altus, pulsus parvus). Volume.

Pulsus altus is observed in febrile states and in association with cardiac hypertrophy of all kinds (especially attending aortic insufficiency).

Pulsus parvus is observed especially in connection with mitral stenosis.

Hardness (dependent upon the tension of the artery).

Pulsus durus occurs in connection with hypertrophy of the left ventricle, with contracted kindey, with lead-colic.

Pulsus mollis is found in febrile states, in connection with mitral stenosis and with anemia.

In feeling the pulse the state of the wall of the vessel is also noted. Of especial importance is the existence of abnormal rigidity, calcification (nodular or beaded arteries), or marked tortuosity of the arteries (radial, brachial, temporal). A conclusion as to the existence of arteriosclerosis of the aorta cannot, however, be reliably based upon demonstrable sclerosis of the peripheral arteries, and *vice versâ.*

Sphygmography (pulse-tracing).

By means of the sphygmograph the pulse may in part be more thoroughly analyzed (especially its temporal course) and be permanently recorded.

Normal Tracing (Figs. 19 and 20):

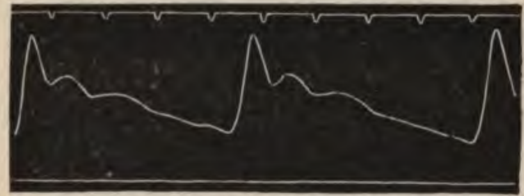


FIG. 19.—Normal pulse-tracing (rapid rotation of the drum).

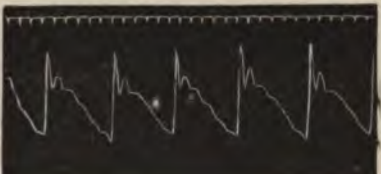


FIG. 20.—Normal pulse-tracing (slow rotation of the drum).

The uninterrupted, oblique ascending line (percussion-wave) is followed by a more slowly descending interrupted line, marked by secondary elevations (tidal or predicrotic wave [*a*], recoil or dicrotic wave [*b*]).[1]

Pulse-tracing of the Febrile State:

With increasing fever, the tension of the vessel-wall diminishes, and the elevation due to recoil of the blood becomes very marked and palpable (*dicrotism*), while the elevations due to the elasticity of the blood-vessel disappear. Figs. 21, 22, and 23 show the alterations in the pulse during an attack of intermittent fever.

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[1] The notches above the tracing are made by the chronometer (5 per second).

At the beginning of the attack at 6 P.M. the pulse-tracing exhibited no peculiarity.

At 11 P.M. the temperature was 39.9° C. (103.8° F.), the pulse 128 and distinctly dicrotic. The tracing (Fig. 21) shows a distinct elevation in the descending line (*catadicrotism*).

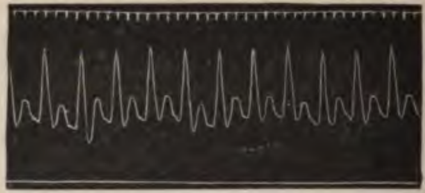

FIG. 21.

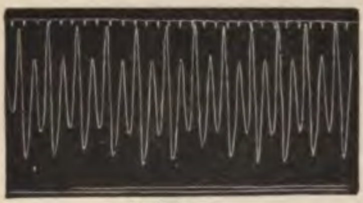

FIG. 22.

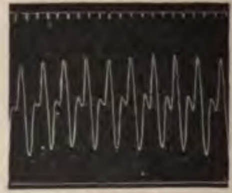

FIG. 23.

At 12.30 A.M. the temperature was 40.6° C. (105° F.), the pulse 146 and markedly dicrotic. The tracing (Fig. 22) shows a marked elevation between the descending and the ascending line (*dicrotism*).

At 1.30 A.M. the temperature was 41.3° C. (106.3° F.), the pulse 160. The tracing (Fig. 23) shows an elevation in the ascending line (*anadicrotism*). A similar condition is illustrated in Fig. 24 (dicrotism in typhoid fever).

Finally, when the action of the heart is excessively rapid the elevation due to the wave of recoil may coincide with the elevation due to the subsequent percussion-wave, so that the pulse becomes *monocrotic*.

When, on the other hand, the tension of the artery is abnormally great, the elevations due to the elasticity of the vessel become more numerous and more distinct.

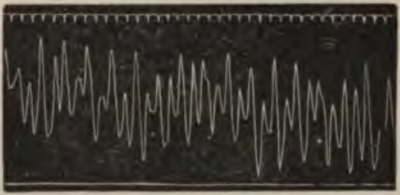

FIG. 24.

Pulsus parvus, pulsus irregularis (mitral stenosis) : Fig. 25. (Slight elevation and irregularity of the pulse-tracing.)

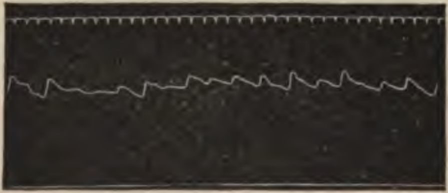

FIG. 25.

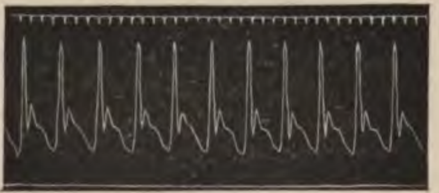

FIG. 26.

Pulsus celer (aortic insufficiency): Fig. 26. (Steep ascent, rapid fall of the percussion-wave.)

Pulsus tardus (aortic stenosis): Fig. 27. (The ascending line is small, the apex blunt, the descending line prolonged and scarcely interrupted.)

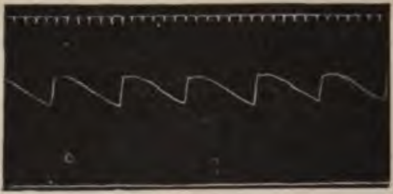

FIG. 27.

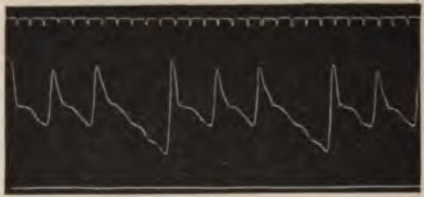

FIG. 28.

Pulsus irregularis (arteriosclerois of the coronary arteries): Fig. 28. (Omission of every third pulse-beat.)

3. The Blood (Plates 1 to 6).

Arterial blood is bright red, in consequence of the presence of oxyhemoglobin, while *venous blood* is dark red in color. The specific gravity of the blood varies between 1050 and 1060. The blood consists of blood-plasma (serum and fibrin) and of the morphotic elements (red and white blood-corpuscles, blood-plates).

The **red corpuscles (erythrocytes)** are the oxygen-carriers (oxyhemoglobin) of the blood, and in healthy men are present to the number of about 5,000,000 to the cu. mm., and in women to the number of about 4,500,000. The average diameter of the red blood-corpuscles is 7.6 μ (6.5–9.3 μ). The erythrocytes exhibit the well-known disc-shape, with a central depression. They are unprovided with nuclei and in fresh preparations exhibit a tendency to form rouleaux. They are very sensitive structures, swelling up in water and losing the coloring-matter (hemoglobin) contained within their stroma. When dried or acted upon by certain reagents (solution of urea, etc.) they become crenated and take on thornapple-forms.

The hemoglobin consists of an albuminous body (globulin) and an iron-containing pigment, known as hematin. Hematin chlorid (hemin) crystallizes in characteristic brownish rhombic plates (Plate 6, Fig. 3).

The **white corpuscles (leukocytes)** are present in the blood to the number of about 7500 (6000 to 9000) to the cu. mm. The majority exhibit active ameboid movement upon a warmed stage. They are colorless, distinctly granular, and possess nuclei that become especially distinct upon addition of acetic acid and upon staining. They vary in size from 5 to 15 μ.

In accordance with the character of their nuclei the following varieties of white corpuscles are distinguished:

1. *Large polynuclear* (with several lobulated nuclei; from 65 to 70 per cent.).

2. *Small mononuclear* (with a single nucleus; about 25 per cent.). A large and a small form of mononuclear leukocyte can be distinguished.

3. *Transitional forms* (leukocytes in process of degeneration; about 5 per cent.).

The white blood-corpuscles contain within their protoplasm small bodies (granulations), which can be differentiated in accordance with their size, and especially in accordance with their affinity for certain stains (acid or basic aniline colors):

1. *Acidophile, Eosinophile Cells.*—These contain coarse granules (α-granules), which are stained a bright red with eosin. They represent about from 1 to 5 per cent. of the leukocytes and are polynuclear cells.

2. *Basophile Cells.*—These contain either

a. Coarse granules (γ-granules; mast-cells), or

b. Fine granules (δ-granules).

Both varieties of granules stain deeply with methylene-blue, etc. The majority of mononuclear cells contain such granules (20 to 25 per cent.).

3. *Neutrophile Cells.*—These contain numerous dust-like granules (ε-granules), which stain violet with solutions of neutral stains. These granules are contained within the polynuclear cells (70 per cent.).

Whether or not a single cell may contain several varieties of granules (*amphophile cells*) is yet a matter of doubt. The nature and the significance of the granules (albuminous bodies) have likewise not yet been determined. For further details, see Plate 2.

Blood-plates.—These occur in grape-like (sometimes also in cylindric) arrangement in masses to the number of about 200,000 to the cu. mm. They appear to stand in a certain relation to the leukocytes with regard to coagulation of the fibrin.

With regard to the *genesis* of the red and the white blood-corpuscles, it is only known with certainty that they are formed in the bone-marrow, in the spleen, and in the lymphatic glands. The erythrocytes develop in the red bone-marrow from nucleated cells (erythroblasts); the mononuclear leukocytes apparently in part in the lymphatic glands; the remainder in part in the spleen. Existing knowledge upon this subject, however, is yet contradictory.

Methods of Examination and their Results.

1. Spectroscopy of the Blood (see Plate 6).

The examination can be satisfactorily made with the hand-spectroscope in place of the large spectroscope. From three to five drops of blood are placed in a test-tube containing water, which is then held between the opening of the spectroscope and a source of light.

The normal blood-spectrum shows between the absorption-lines D and E two black bands (oxyhemoglobin). If the solution of blood be reduced by addition of several drops of a solution of ammonium sulphid or copper sulphate, these two bands disappear, and are replaced by a new wider band (reduced hemoglobin).

In cases of carbon-monoxid poisoning the oxyhemoglobin is converted into CO-hemoglobin, whose spectrum corresponds in the main with that of the former. If reduction be effected, however, the two absorption-bands do not disappear (differentiation from oxyhemoglobin-spectrum; stronger combination of CO).

In certain cases of hemoglobinemia (disintegration of erythrocytes in the blood), following poisoning with potassic chlorate, etc., methemoglobin is formed in the blood. The spectrum then shows between A and *α* a small intense band, while the spectrum beyond green is completely absorbed. (See Plate 6, Fig. 1, *d*.)

2. Estimation of the Hemoglobin.

The hemoglobin is most conveniently estimated by means of the apparatus of Gowers. Hermetically sealed in a small glass tube is a solution of red color (carmine-glycerin), corresponding in its intensity with a 1 per cent. solution of normal blood. With this normal solution is to be compared the blood to be studied. To 20 cu. mm. of the latter, carefully taken up with a pipet, water is added successively until the intensity of the red color corresponds with that of the normal solution. In making the comparison the tubes are held in front of white paper, and the percentage of hemoglobin is read from the accompanying scale (error of about 5 per cent.).

Diminution in the amount of hemoglobin, *oligochromemia*, may fall as low as 15 per cent. of the normal, and occurs in connection with chlorosis and all forms of anemia. The amount is *increased* in connection with pulmonary stenosis.

Normally, 100 cu. cm. of blood contain from 13 to 14 grams of hemoglobin.

3. Enumeration of the Red and White Blood-corpuscles.

For this purpose the apparatus of Thoma-Zeiss is employed.

In an absolutely dry capillary tube (washed with alcohol and ether) is drawn up 0.5 (or 1) cu. mm. of blood, which is diluted with water to the mark 101. The two are then well intermixed, and a drop is placed in the chamber of a suitably constructed slide, the presence of air-bubbles being obviated. Of the large squares scratched on the slide (each containing sixteen small squares) the contents of not less than fourteen are counted, and the result is determined by the following formula:

$\frac{x \, . \, 200 \, . \, 4000}{14 \, . \, 16}$, x = the number of cells counted,

$\frac{1}{4000}$ cu. mm. = the area of a small square.

If 1 cu. mm. of blood has been used, the multiple should be 100, instead of 200.

The enumeration of the white blood-corpuscles is made with a larger capillary tube, and a 1 per cent. solution of acetic acid is used (to destroy the red blood-corpuscles) as a diluent, instead of water. The other steps in the procedure are the same as those for enumeration of the red cells.

Diminution in the number of red blood-corpuscles (oligocythemia) occurs (as low as 500,000) in connection with pernicious anemia, the secondary anemias (not with chlorosis), leukemia, profound loss of blood.

Increase in the number of white blood-corpuscles occurs—

1. As a transient manifestation (leukocytosis): physiologically (about 10,000 to 12,000) after digestion, in the new-born, during pregnancy; pathologically (to 60,000), in a number of infectious diseases (pneumonia, purulent meningitis, erysipelas, scarlet fever, diphtheria, polyarthritis, pyemia).

Further, leukocytosis is not rarely found, but not with absolute regularity, in cases of chlorosis, in cachectic states (carcinoma, hydremia), in the death-agony.

The increase under these conditions affects especially the ~~polynuc~~lear cells with ameboid movement. The eosinophile cells are regularly present in but small numbers in inflammatory states, but they are increased under conditions not yet precisely determined in some forms of anemia, in cases of asthma, etc.

2. Permanent increase in the number of leukocytes occurs in conjunction with leukemia (50,000 to 300,000 in the cu. mm.).

For the establishment of the diagnosis of leukemia marked increase in the number of leukocytes (which exhibits wide variations even in individual cases) alone is, however, not sufficient, but it must be supplemented by the results of further clinical investigation, especially microscopic examination.

As the number of erythrocytes is almost constantly diminished in cases of leukemia, the normal relation between the red and the white blood-corpuscles may be reduced as low as 2 to 1, instead of the normal 700 to 1.

4. Microscopic Examination of the Blood.

A puncture with a sharp, triangular needle is quickly made in the carefully cleansed and dried finger-tip, the first drop of blood escaping being removed, and the second being taken upon an absolutely dry cover-glass (washed with alcohol and ether). Upon this is placed a second cover-glass, and the two are separated, with slight pressure, by means of forceps. Especial care is to be taken to prevent the access of moisture to the cover-slips in order to avoid destruction of the erythrocytes through the presence of water. With the prepared surface upward the preparation is permitted to dry in the air (for half an hour).

In the meantime the fresh blood can be examined, a cover-glass with a drop of blood being gently pressed upon a slide. With partial illumination attention will be directed to—

1. *The Erythrocytes.*

a. The *size* of the red blood-corpuscles (measurement with the ocular micrometer).

Abnormally small erythrocytes, microcytes, from 2 to 6 μ in diameter, are found in cases of anemia, chlorosis, and leukemia.

Abnormally large erythrocytes, macrocytes, from 10 to 15 μ in diameter, are found under like conditions.

b. The Form.—Changes in form are often of artificial nature, but occurring extensively and in marked degree they justify a conclusion as to abnormally increased lability in the consistence of the erythrocytes.

In cases of profound anemia the most varied alterations in form are frequently found (protrusions, indentations, serrations, pear-shaped, kidney-shaped appearances). This condition is designated poikilocytosis.

c. The Color.—The normal yellowish-red hue of the blood may be replaced by a paler appearance, especially in cases of chlorosis.

d. The tendency of the blood-cells to form rouleaux is diminished in cases of anemia.

2. *The Leukocytes.*

In unstained preparations an approximate determination of the number of white blood-corpuscles is possible. With a magnification of 500, every five white blood-corpuscles in the field of the microscope correspond to 8000 in the cu.mm. If, therefore, on examination of twenty fields an average of fifteen leukocytes in each be found, it may be estimated that there exists a leukocytosis of about 24,000. The ameboid movement of the white corpuscles may also be observed in unstained preparations. The eosinophile cells are to be recognized by their bright, yellowish, coarse granulation.

Stained Preparations.—The cover-slip, spread with a drop of blood and dried in the air, is fixed by being passed rapidly ten times through the flame of an alcohol lamp or a Bunsen burner, or better, by being heated for two hours at a temperature of 120° C. (248° F.), or by being exposed for ten minutes in a 1 per cent. solution of formol-alcohol, or by exposure for one hour in equal parts of absolute ether and alcohol.

Staining solutions (best obtained from Gruebler, of Leipsic):

1. 1 per cent. watery solution of eosin.
2. Solution of methylene-blue and eosin. (See p. 22.)
3. Eosin, aurantia and nigrosin, of each 2., glycerin 30.

4. Ehrlich's solution of hematoxylin and eosin. (See p. 21.)

5. Ehrlich's triacid solution (contains a basic, an acid, and a neutral aniline stain).

6. Ehrlich's neutral stain (containing acid fuchsin and methylene-blue).

It is best next to proceed with the staining of a fixed preparation with methylene-blue and eosin, the specimen being exposed for ten minutes, with heat. The remaining preparations are treated for twenty-four hours with the solution of eosin, aurantia, and nigrosin, and with Ehrlich's solution of hematoxylin and eosin. They are then washed, dried in the air, and mounted in Canada balsam. With Ehrlich's triacid solution and Ehrlich's neutral solution the exposure should be about five minutes.

The stained preparations exhibit well the nuclei and the forms of the blood-corpuscles, the granulations of the leukocytes, and the presence of possible parasites. Only those who have made themselves familiar with stained preparations can be depended upon to recognize the varied conditions in fresh preparations.

Concerning the individual peculiarities to be observed in stained preparations, as well as their significance, reference should be made to Plates 1 to 6, and the accompanying text.

III. EXAMINATION OF THE DIGESTIVE APPARATUS.

I. Mouth and Pharynx.

Lips.

There may be present eruptions of herpes (frequently accompanying pneumonia, meningitis, malaria, etc.), cyanosis (diseases of the heart and of the lungs), marked pallor (anemia), dryness, fissures (attending fever), brownish, fuliginous deposit (typhoid fever).

Teeth and Gums.

The *deciduous teeth* appear in pairs on an average every two months from between the fifth and the eighth month of life, the lower usually in advance of the corresponding upper pairs, and in the following order:

From the eighth to the twelfth month, the median incisors;

From the twelfth to the sixteenth month, the lateral incisors;

From the sixteenth to the twentieth month, the anterior molars;

From the twentieth to the twenty-fourth month the four canine;

From the twenty-fourth to the thirtieth month, the posterior molars.

The *permanent teeth* begin to make their appearance at the seventh year in much the same order, four appearing each year in the place of the exfoliated deciduous teeth. The first bicuspid appears in the ninth, the second bicuspid in the tenth year. Three molars complete the set, the last of which may not appear before the third decade.

Imperfect teeth interfere with normal digestion (deficient mastication and insalivation).

Stomatitis, gingivitis (swelling, redness, crust-formation, and ulceration) attend mercurial poisoning, scorbutus, and aphthæ. A *blue line* (blackish deposit of lead sulphate at the margin of the gums) occurs as a result of lead-poisoning.

Tongue.

A coated tongue (epithelial desquamation, remnants of food, bacteria) attends gastric catarrh; a smooth moist tongue, hyperacidity; a smooth tremulous tongue, chronic alcoholism; a dry, brownish, fissured tongue, febrile states; a leathery tongue, coma and collapse; the "strawberry" tongue, scarlet fever.

Palate and Tonsils.

Attention is to be directed to abnormal redness and swelling (catarrhal angina, abscess-formation, scarlet fever).

Deposits attend follicular angina (isolated, disseminated whitish plugs of pus), diphthéria (coherent, grayish-white coating consisting of fibrin, pus, and bacteria, and leaving a bleeding surface when removed with forceps), thrush-vegetations (fungous masses of oïdium albicans observed in children and in comatose states).

Ulceration and Perforation of the Palate: adhesions to the pharynx in cases of syphilis.

Pharynx.

Chronic Pharyngitis (attending alcoholism, occurring in smokers, singers, and those who work in dust) is characterized by a smooth, shining atrophic appearance of the mucous membrane, with swollen follicles and the formation of tough secretion.

Retropharyngeal Abscess (visible and palpable prominence of the posterior wall of the pharynx) should suggest itself when symptoms of profound septic infection are present, in conjunction with difficulty in swallowing and in breathing. It occurs in the sequence of glandular and vertebral disease, especially in children.

For the microscopy of the buccal cavity, see Plate 7.

2. Esophagus.

The esophagus is 25 cm. (9¾ in.) long; the distance from the margin of the teeth to the beginning of the esophagus is additionally 15 cm. (6 in.); so that the distance from the margin of the teeth to the cardia is 40 cm. (15¾ in.). The distance from the margin of the teeth to the intersection of the esophagus by the left bronchus is 23 cm.—9 in. (important in examination with a sound).

In making examinations with a sound (for the method, see p. 29) it is best to employ a hard, solid, or preferably a soft, hollow instrument. The use of the sound is contraindicated when aneurism of the aorta is suspected, in order to avoid possible perforation. If the esophagus be patulous, the sound passes readily into the stomach (with certainty if it can be introduced beyond a distance of 40 cm.—15¾ in.).

In cases of *narrowing of the esophagus* obstruction to the passage of a sound is encountered at a corresponding level. An obstruction at a distance of 23 cm. (9 in.) from the margin of the teeth will be at the level of the junction of the esophagus with the left bronchus (diverticula, glandular swellings, aneurism, cicatrices); one at a distance of from 38 to 40 cm. (15–15$\frac{3}{4}$ in.) at the level of the cardia (most frequently carcinoma).

Esophageal diverticula (saccular dilatations) may be seated at the beginning of the esophagus (pulsion-diverticula). When filled with food they may compress the esophagus and be visible through the neck as distinct swellings. They may also be more deeply seated (traction-diverticula), resulting from contraction of adjacent glands. They may give rise to abnormal communications between esophagus, bronchi, and large vessels, but they are not accessible to accurate diagnosis, being frequently found accidentally upon postmortem examination.

Above the point of narrowing there is usually a secondary dilatation of the esophagus.

The vomiting attending esophageal stenosis is characteristic. It takes place often directly after the ingestion of food, and the patient is conscious of a sense of stoppage.

The swallowing sound (see p. 34) may be wanting or delayed when narrowing of the esophagus exists.

3. Stomach.

Anatomic.

The stomach lies for five-sixths of its extent in the left half of the body, and for its remaining one-sixth in the right half. The fundus lies in the concavity of the left half of the diaphragm. The greater curvature, the lower limit of the stomach, passes from 2 to 4 cm. ($\frac{3}{4}$–1$\frac{1}{2}$ in.) above the umbilicus; the lesser curvature is covered by the margin of the liver, as is also the pylorus, which lies in the right sternal line at the level of the ninth costochondral junction (sometimes higher, sometimes lower in individual cases). That portion of the stomach which lies in contact with the anterior and lower portion of the left half of the thorax gives rise to the tympanitic note of Traube's semilunar space. The boundaries of this space are, therefore, the left lower margin of the lung (above); the left border of the liver (on the right); the splenic dulness (on the left); the left costal arch (below). (See Plate 24.)

Physiologic.

When the masticated bolus reaches the stomach gastric digestion sets in, with the secretion of hydrochloric acid and pepsin (proferment pepsinogen). As a result the albuminous bodies are converted into acid albuminates through combination with hydrochloric acid, and with the simultaneous action of the pepsin they are transformed, through intermediate stages (albumoses), into peptone-like bodies in part soluble.

So-called *free hydrochloric acid* (not in combination with albuminous bodies) is distinctly demonstrable half an hour after the ingestion of a meal.

The *total acidity* (free and combined hydrochloric acid) equals from 1.5 to 2 per cent. one hour after the ingestion of food. In addition to hydrochloric acid, other (organic) acids are formed in the process of gastric digestion. These develop as a result of fermentation (bacterial activity) from starch and sugar: lactic acid, acetic acid, butyric acid. The acid fermentation is related to the production of hydrochloric acid. The more acid present the less is the amount of organic acids (antagonism).

In accordance with the amount and the character of the meal the stomach gradually empties its contents into the bowel by peristaltic contractions. An ordinary mid-day meal should have disappeared from the normal stomach after six hours. The fasting healthy stomach should therefore be empty early in the morning.

Deviations from the normal process of gastric digestion consist—

1. In variations in the amount of hydrochloric acid produced. There may be too much (hyperacidity), or too little (anacidity).

2. In the amount of organic acids formed (abnormal in association with anacidity).

3. In derangement of the act of evacuation of the stomach. Retarded emptying of the stomach (motor insufficiency) occurs in conjunction with diminished physical vigor, or with abnormally increased resistance (pyloric stenosis).

Inspection.

The stomach may be made visible by distention with gas. Especially in marked cases of dilatation may the entire contour of the viscus become distinctly visible. It is important in this connection to recognize the peristaltic movements of the stomach, which take place from the left above to the right below, and may be elicited by gentle tapping.

Palpation.

Strictly circumscribed severe pain in the epigastric region, increased upon pressure, is suggestive of ulcer of the stomach.

Tumors in the region of the stomach, especially of the greater curvature (carcinoma), may be distinctly felt in the region of the umbilicus when the abdominal walls are relaxed (if necessary, anesthesia may be induced). Tumors of the stomach, in contrast with those of the liver, are but little movable with respiration.

In the region of the pylorus there occur, in addition to carcinoma, hypertrophic cicatrix-formations (associated with hyperacidity), which may be palpable as small tumors.

For description of splashing-sounds, see p. 34. They are of diagnostic significance only in conjunction with other symptoms.

Percussion.

This yields reliable results only in combination with other methods of investigation (filling the stomach with air or with water). Under these circumstances there can be determined the situation of the lower boundary of the stomach and the existence of possible dilatation. If this lower boundary is situated at or below the level of the umbilicus, it may be concluded that dilatation of the stomach exists, providing that enteroptosis be not present—that is, an abnormally low position of the abdominal viscera.

The stomach can be distended with gas by the administration of a spoonful of sodium bicarbonate followed by a spoonful of tartaric acid. The carbonic acid evolved causes distention of the stomach, and the deep, full percussion-note of the stomach is distinctly separable from the higher note yielded by the intestine. By introducing the stomach-tube air may be injected directly, or by means of irrigation with the tube the boundaries of the stomach may be determined by percussion, the resulting area of dulness corresponding with the lowest portion of the viscus. Frequently the distention of the stomach thus produced is also distinctly visible.

The Study of Gastric Digestion.

It is important to determine accurately the secretion of hydrochloric acid, the presence of organic acids, and the motor activity of the stomach. (a dig. of alb. - pepsi

1. *Qualitative determination of free hydrochloric acid.* (See Plate 12.) If the gastric juice contain free hydrochloric acid,

a. Blue litmus-paper becomes red on immersion;

b. Red congo-paper becomes intensely bluish-black;

c. Dilute solution of methyl-violet is changed in color from violet to blue on addition of several drops of the gastric juice;

d. On mixing several drops of Günzburg's reagent (phloroglucin 2.0, vanillin 1.0, absolute alcohol 30.0) with several drops of the gastric juice in a porcelain dish a distinct red band results on evaporation.

The last test (*d*) is the most reliable; it will detect the presence of hydrochloric acid even when in very great dilution (as little as 0.05 per cent.). Tests *b* and *c* may yield positive reactions also in the presence of organic acids; test *a* also in the presence of acid salts (phosphates).

2. *Qualitative determination of lactic acid.*

Of organic acids, lactic acid is the most important. Its detection is effected by means of Uffelmann's reagent (to 10 cu. cm. of a 1 per cent. solution of carbolic acid are added two drops of iron-chlorid solution), which is always to be prepared fresh. The original bluish-violet tint of the reagent becomes yellow on the addition of gastric juice containing lactic acid. The test is more reliable if the lactic acid be isolated from the gastric juice by agitation with ether, and the extract is used in making the reaction.

Quantitative determination of the total acidity.

The quantitative determination of hydrochloric acid alone is difficult and time-consuming. For clinical purposes, however, it suffices to determine the total acidity of the gastric juice resulting from the combination of free hydrochloric acid, free organic acids, and acid salts. The result, estimated as hydrochloric acid, will naturally be somewhat too large.

In making the determination 10 cu. cm. of unfiltered gastric juice are titrated with $\frac{1}{10}$ normal soda-solution. Two drops of an alcoholic solution of phenolphthalein are added as an indicator to the measured amount of gastric juice diluted with water until colorless. The soda-solution is allowed carefully to drip from the pipet, the reaction being terminated when the mixture retains a faint rose hue.

If 5 cu. cm. of the soda-solution have been needed for 10 cu. cm. of gastric juice, 100 cu. cm. of the latter would have required 50 cu. cm. of the soda-solution, so that the total acidity would be 50 per cent.; 1 cu. cm. of the normal soda-solution contains 0.004 g. of sodium hydroxid and 50 cu. cm. 0.2. The amount of hydrochloric acid is determined according to the following formula:

$$HCl = \frac{36.5 \times 0.2}{40} = 0.182 \text{ per cent.}$$

The course of examination may proceed as follows:

3. Motor activity.

1. *The stomach is washed out early in the morning, fasting.*—If on introduction of the tube nothing can be expressed from the stomach (with the co-operation of the patient), and if upon irrigation of the stomach with warm water only this is returned, it can be concluded that the stomach is normally empty. If the patient by pressure expels fluid after the introduction of the tube, there exists hypersecretion, and examination for hydrochloric acid is made. If this be distinctly demonstrable, there exists also hyperacidity.

Hypersecretion with hyperacidity occurs in conjunction with ulceration of the stomach and with nervous acid dyspepsia.

If the stomach contain remnants of food, there exists motor insufficiency.

2. *The trial-breakfast.*—This consists of one cup of tea and a roll. From one-half to three-quarters of an hour after ingestion the gastric contents are expressed through the tube and the filtrate is examined for hydrochloric and lactic acids.

The filtrate should normally respond to all of the tests for hydrochloric acid. Especially must the phloroglucin-vanillin test yield a positive reaction; otherwise there exists anacidity.

In this event the test for lactic acid will yield a positive reaction. A pronounced lactic-acid reaction (after previous thorough irrigation of the stomach) is suggestive, as a rule, of carcinoma.

Anacidity may exist in conjunction with chronic gastric catarrh, with atrophy of the gastric mucous membrane, with anemia, and especially with carcinoma. If an active reaction be yielded to the tests for hydrochloric acid, carcinoma can be excluded with considerable certainty.

If the stomach has been thoroughly washed out before the trial-breakfast is given, removing possible stagnant organic acids, the unfiltered gastric contents can be employed for the determination of the total acidity, and from the amount of the estimated hydrochloric acid a conclusion can be reached as to the existence of possible hyperacidity.

3. *The trial-meal.*—This consists of beefsteak (200 to 250 g.). After the lapse of six hours the stomach should be found completely empty on irrigation; otherwise motor insufficiency exists.

ʒ 6-

The contraindications to washing out the stomach are grave diseases of lungs and heart, recent hemorrhage from the stomach, aneurism of the aorta.

For further investigation of the motor activity of the stomach the salol-test among others may be employed. With the meal 2 g. of salol are given in wafer. The salol passes through the stomach without decomposition and is broken up in the alkaline contents of the intestine into salicylic acid and phenol. If the motor activity be normal the former appears in the urine in from three-quarters of an hour to an hour, a violet color developing upon the addition of iron chlorid. In case of motor insufficiency the reaction will not take place earlier than after the lapse of from two to five hours, and it may persist for two days.

The capability of digesting albumin is tested by exposing flakes of fibrin to the action of the filtered gastric juice. Normally solution takes place in from six to eight hours. If this have not taken place in the period named in a warm temperature, while solution occurs upon the addition of several drops of 1 per cent. hydrochloric acid, it is to be concluded that hydrochloric acid is absent. If even now the fibrin remains undissolved, pepsin is absent.

Through the action of the labferment (pexin) of the normal stomach fresh milk is coagulated in from one-quarter to one-half of an hour on the addition of several drops of filtered gastric juice.

Vomiting.

Vomiting results from antiperistaltic contractions of the stomach, with simultaneous contraction of the diaphragm and the abdominal muscles, and closure of the pylorus and opening of the cardia.

The act of vomiting follows direct or reflex irritation of the vomiting-center in the medulla oblongata:

Directly through the action of emetics, poisons (chloroform), toxic diseases (uremia, fever);

Reflexly from the stomach (ulceration, carcinoma, catarrh, etc.), from the intestines and the peritoneum (stenosis of the bowel, peritonitis), in conjunction with gravidity, diseases of the brain, etc.

Periodic vomiting occurs in cases of cholelithiasis (in conjunction with attacks of colic), of tabes dorsalis (gastric crises), of neurasthenia.

Repeated vomiting of large amounts is characteristic of dilatation of the stomach.

Vomiting of blood (hematemesis) occurs in conjunction with ulceration of the stomach (fresh, dark-red blood), with carcinoma (disintegrated coffee-ground blood), with erosion (acids, alkalies), vicariously in connection with anomalies of menstruation.

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Expectorated blood (hemoptysis) is bright red and frothy if it have not been previously swallowed and vomited subsequently.

Fecal vomiting occurs in conjunction with occlusion of the bowel (incarceration, intussusception, volvulus).

Morning vomiting on an empty stomach is indicative of chronic pharyngeal catarrh (sensitive mucous membrane), and is common in alcoholics.

Constitution of the vomited matters.—These contain particles of food in process of digestion and of fermentation, albuminous bodies (albumoses, peptone, leucin, tyrosin), starch, carbohydrates (through fermentation yielding lactic acid butyric acid, acetic acid, valerianic acid), fat and fatty acids, coagulated milk, swallowed saliva (yielding a red color on addition of iron chlorid in consequence of the presence of potassium rhodanate), mucus (from esophagus and stomach), bile (from the duodenum, especially in connection with severe nausea), urea (uremia).

For the microscopy of the vomited matters Plate 11, Fig. 1, may be consulted.

4. Liver.

The liver lies three-quarters in the right and only one-quarter in the left half of the body. Its convex surface lies in close approximation with the diaphragm. The organ is entirely covered with peritoneum.

Its sharp, lower border is situated at about the level of the eleventh rib in the axillary line on the right side; it intersects the costal arch (ninth costal cartilage) in the mammillary line; it passes midway between the umbilicus and the root of the ensiform cartilage in the median line; and is at the level of the eighth costal cartilage in the left parasternal line. (See Plate 23.)

The gall-bladder lies exactly at the point where the liver emerges from beneath the right half of the costal arch—that is, at the level of the ninth costal cartilage somewhat within the mammillary line. In children the liver is relatively larger than in adults.

The liver follows all movements of the diaphragm (respiration, elevation and depression).

The most important functions of the liver are:

a. The elaboration and secretion of bile (digestion of fats);

b. The conversion of the sugar of the food into glycogen;

c. The disposition of disintegrated red corpuscles, of toxic substances, etc.

The most important symptoms of disease of the liver are jaundice and ascites.

Jaundice (deposition of biliary coloring-matter in the skin, sclera, etc.) results in consequence of the passage of the biliary constituents into the blood, in conjunction with occlusion of the biliary passages (choledoch duct, hepatic duct, biliary capillaries), resulting from catarrh, from the formation of calculi, from the presence of tumors (carcinoma), as well as in conjunction with certain infectious diseases (enormous destruction of erythrocytes in the liver). The pulse is slow (influence of the biliary acids).

If the access of bile to the intestine is prevented, the digestion of fats is rendered difficult, and the stools become acholic (free from biliary coloring-matter), grayish-white and clay-colored, and they contain many fat-crystals. Some of the bile circulating in the blood is excreted with the urine, which as a result becomes dark brown in color, and on shaking forms a yellowish foam (for the reactions see Section III., p. 112).

Simple jaundice results from catarrhal swelling of the choledoch duct in conjunction with duodenal catarrh, and is of short duration (from two to four or six weeks).

Grave jaundice (of longer duration, attended with nutritive disturbances, pain, chills) occurs in conjunction with cholelithiasis (repeated attacks of colic, vomiting), with carcinoma of the liver or biliary passages (cachexia, tumors of the liver), with cirrhosis of the liver (associated with ascites), with abscess of the liver (fever, chills). Amyloid liver, fatty liver, and echinococcus also are usually unattended with jaundice.

Ascites (dropsy of the peritoneum) occurs in conjunction with pressure upon the portal vein or embarrassment of the circulation of blood in the liver-capillaries in consequence of stasis in the veins of the peritoneum, the omentum, the stomach, etc., with closure and with compression of the portal vein (tumors, thrombosis), with cirrhosis of the liver, rarely with syphilis and carcinoma of the liver. The spleen is enlarged simultaneously (cyanotic spleen). Ascites due to disease of the liver is not attended with edema of the lower extremities (in contrast with general stasis from disease of the heart). Edema of the lower extremities may, however, appear subsequently (in consequence of compression of the abdominal veins).

Palpation.

The normal liver is not palpable below the costal arch in the mammillary line. The lower border of the liver becomes palpable—

When the liver is displaced downward (pleurisy, pneumothorax, tumors) and also when a wandering liver is present ;

When the liver is enlarged, or constricted (movable lobe, fissure), or cyanotic, or fatty, or amyloid;

When hypertrophic cirrhosis, carcinoma, syphilis, or abscess exists.

The palpable margin of the liver may be—

Hard and smooth: cyanotic liver, amyloid liver (blunt), hypertrophic cirrhosis, echinococcus; softer: fatty liver;

Irregular, nodular: cirrhosis, carcinoma, syphilis, and at times with (superficial) abscesses.

Percussion.—*Normal liver-dulness:*

The upper limit in the mammillary line corresponds with the lower border of the sixth rib; the lower limit coincides with the lower border (see anatomic notes).

Diminution in the area of hepatic percussion-dulness occurs in conjunction—

1. With respiration (on deep inspiration the upper limit is displaced downward); with emphysema, as a result of overlapping of the lung;
2. With meteorism, overlapping of the intestines, ascites, abdominal tumors;
3. With the entrance of air into the abdominal cavity (perforative peritonitis);
4. With cirrhosis of the liver;
5. With acute yellow atrophy of the liver.

Increase in the area of hepatic percussion-dulness is observed in conjunction with cyanotic liver, hypertrophic cirrhosis, amyloid liver, fatty liver, carcinoma, echinococcus, abscess-formation.

The enlarged gall-bladder can be demonstrated by palpation and by percussion below the ninth costal cartilage in conjunction with an accumulation of calculi (nodular), with carcinoma (uneven, solid tumor), with hydrops from chronic occlusion by calculus-formation or from obliteration (smooth, tense).

5. Spleen.

The spleen is situated in the left hypochondrium, reaching from the ninth to the eleventh rib, with its posterior boundary about 2 cm. from the body of the tenth dorsal vertebra. Anteriorly it does not extend beyond the costo-articular line (from the left sterno-clavicular articulation to the apex of the eleventh rib). The area of splenic dulness corresponds with these boundaries. The normal spleen is not palpable, and when it becomes so it may be concluded that it is either displaced or enlarged.

Palpation of the spleen is practised by having the patient lie upon the right side, with the thighs drawn up, the ulnar border of the hand being gradually introduced beneath the left half of the costal arch. By this means it may be possible, especially on deep inspiration, to feel a solid, resisting body.

The area of splenic percussion-dulness is determined by percussing first from above downward parallel with the left intercostal spaces (thus obliquely downward) until dulness is reached. The finger is now placed at right angles to its former position, and percussion is practised in a forward direction until a tympanitic note is yielded (the bowels having been previously emptied).

Displacement of the spleen takes place in conjunction with pleurisy and pneumothorax (downward), with meteorism, ascites, abdominal tumors, and wandering spleen.

Enlargement of the spleen takes place in conjunction with infectious diseases (typhoid, malaria, pyemia, etc.), amyloid degeneration, cirrhosis of the liver, stasis in the general circulation (cyanotic spleen), hemorrhagic infarction (embolism) of the spleen, leukemia (frequently distinct indentations are palpable).

Abolition of splenic dulness occurs in conjunction with perforative peritonitis (accumulation of air over the spleen in right lateral decubitus).

6. Abdomen, Intestines, and Peritoneum.

In health the abdomen is slightly convex, takes part in the rhythmic movements of respiration, and yields a tympanitic note on percussion (slight dulness only over considerable fecal accumulations in the large intestine).

Abnormal retraction of the abdominal walls takes place in conjunction with intestinal colic (lead-colic), meningitis (scaphoid abdomen), and marked emaciation (carcinoma).

Distention of the abdomen occurs in conjunction with:

1. *Meteorism* (accumulation of gas in the intestines). The abdomen is uniformly balloon-like-shaped and yields everywhere a loud, high note, without fluctuation. Meteorism attends obstinate constipation, abnormal fermentative processes (intestinal catarrh), typhoid fever. It is especially marked in conjunction with stenosis and occlusion of the bowel, ileus (incarcerated hernia, invagination, torsion of the bowel), in association with fecal vomiting and collapse; further with acute peritonitis (only bilious, but not feculent vomiting, tenderness, collapse).

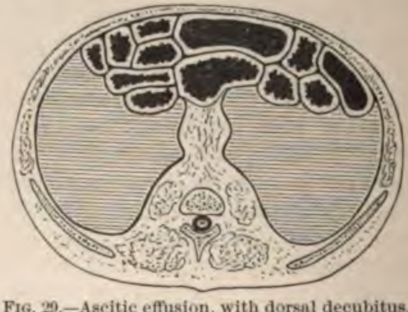


FIG. 29.—Ascitic effusion, with dorsal decubitus.

2. *Ascites* (accumulation of fluid free in the abdominal cavity).

The distention affects especially the lateral aspects of the abdomen (bulging). Absolute dulness is present, in accordance with the posture (see Figs. 29 and 30), always in the dependent portions (displacement of the fluid by gravity); the upper boundary of the dulness is crescentic. The intestines are displaced to the upper portion of the abdomen, where they yield a clear tympanitic note. If the ascites be excessive, the intestines may be held down by their mesenteric attachment so as nowhere to reach the abdominal wall, and the dulness on percussion thus becomes complete. If the ascites be but slight, dulness appears in the region of the umbilicus when the prone position is assumed.

The fluid in the abdominal cavity yields a distinct sense of fluctuation upon palpation (wavy movement). Exploratory puncture is always indicated for confirmation.

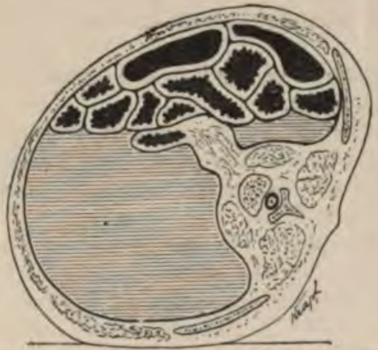


FIG. 30.—Ascitic effusion, with lateral decubitus.

Ascites due to stasis occurs in conjunction with:

1. Diseases of the portal vein and of the liver (stasis in the portal circulation, the peritoneal veins, etc.). At the same time the spleen is enlarged.

2. General dropsy from derangement of compensation in connection with heart-disease, and associated with edema of the extremities, infiltration of the thoracic and abdominal walls, cyanotic kidney, etc.

The transudate contains little albumin and has a specific gravity of between 1006 and 1015.

Inflammatory ascites occurs in conjunction with—

1. Tuberculous peritonitis;

2. Carcinoma of the peritoneum.

The diagnosis is based upon the demonstration of tuberculosis or carcinoma in other organs (pulmonary tuberculosis, glandular tuberculosis, pleurisy, carcinoma of stomach, rectum, ovary, or uterus).

The extravasated fluid is richer in albumin and has a specific gravity of more than 1018 (to be measured with the areometer at room-temperature).

If the exudate in a case of chronic peritonitis is sacculated, variations in dulness do not take place with changes in posture.

3. The entrance of air into the abdominal cavity (perforative peritonitis).

This may occur in conjunction with perforation of a chronic ulcer of the stomach, with tuberculous, dysenteric, and typhoid ulceration, and with perforation of the vermiform appendix.

The accumulation of air occupies the uppermost position, displacing the liver entirely or largely in the dorsal decubitus (disappearance of the liver-dulness) and the splenic dulness in the right lateral decubitus. Frequently also fluid is demonstrable at the same time in the dependent portions of the abdomen.

4. Tumors. The distention due to the presence of new-growths is usually unsymmetric, as are also the area of dulness on percussion and the phenomena disclosed on palpation. Frequently also ascites is present at the same time.

The tumors may originate from the liver (enlargement; carcinoma, especially metastatic), from the stomach (carcinoma), from the spleen (malaria, leukemia), from the omentum (carcinoma, tuberculosis), from the pancreas (cysts), from the intestine (exudation; perityphlitis, in the cecal region; carcinoma), from the ovaries (cysts), from the uterus (gravidity, myomata, fibromata), from the aorta (aneurism), from the vertebral column (abscess), from the kidneys (sarcoma).

When the existence of a tumor is suspected examination should be made always before and after evacuation of the bowels (scybala are of a doughy consistence and are usually palpable).

The point of origin of a new-formation should be determined, if possible. If cysts or exudates are present, exploratory puncture is important. Ovarian and uterine tumors exhibit (in contrast with ascitic dulness) a convex upper boundary, the dulness being situated in the middle of the abdomen, while the note on either side is clear.

The Stools.

The *normal stool* is of a brownish color (in consequence of reduction of biliary coloring-matter, hydrobilirubin, effected through bacterial activity). The stool of the nursing-infant (milk-stool) is yellowish. The stool contains the undigested elements of the food (especially vegetable residue), together with the remains of unabsorbed digestive fluids (bile, intestinal juice).

The *color* of the stools becomes especially striking in conjunction with—

Jaundice: grayish-white (fatty stool, acholic stool);

Diarrhea: greenish (unchanged biliary coloring-matter);

The administration of *iron and bismuth:* black (sulphur salts);

The administration of *calomel:* greenish-brown (biliary coloring-matter and mercury sulphid);

The admixture of *blood:* blackish-brown, tar-like (when the blood comes from the stomach or the upper portion of the small intestine), in cases of gastric hemorrhage, of embolism of the mesenteric artery, and of typhoid fever; bright red (when the blood comes from the large intestine) in cases of hemorrhoids, of intestinal ulceration, and of dysentery;

Typhoid fever: pea-soup-like;

Asiatic cholera: ricewater-like.

Admixture of *mucus* may occur in conjunction with—

Catarrh of the large intestine (covering the surface of the feces);

Catarrh of the small intestine (intimately admixed with feces, sago-grains);

Catarrh of the duodenum (bilious mucus);

Dysentery (blood-streaked);

Ulceration (purulent), occurring in cases of dysenteric, syphilitic, and tuberculous ulceration;

Membranous enteritis (mucous colic of nervous individuals); occurring as tubular casts of the mucous membrane;

Carcinoma of the rectum (pure or bloody mucus).

Pus is found in the stools in conjunction with—

Rupture of an abscess (paratyphlitis, suppurating cysts, exudates);

Dysentery (sanious);

Ulceration of the rectum (syphilis, tuberculosis, carcinoma).

Calculi are found in conjunction with—

Cholelithiasis (cholesterin, bilirubin, and lime),

Fecal concretions (especially of lime and magnesia).

Constipation occurs in conjunction with—

1. Intestinal atony in cases of neurasthenia, of insufficient bodily exercise, in bed-ridden patients, in cases of habitual obstipation, of opium-addiction, and of pyloric stenosis;
2. Stenosis or occlusion of the bowel (ileus); in the presence of either of these conditions there is no discharge of flatus.

Diarrhea occurs—

1. As a result of nervous influences (rapidly transient excitation of intestinal peristalsis);
2. In conjunction with abnormal fermentative processes in the intestine (cholera morbus, cholerine, cholera) as a result of the development of toxic substances;
3. In conjunction with chronic intestinal catarrh, amyloid disease of the intestine (deficient resorption of nutritive substances);
4. In conjunction with ulcerative processes (typhoid fever, tuberculosis, dysentery, syphilis);
5. In conjunction with circulatory disturbances (stasis in the portal vein, general stasis);
6. In conjunction with tabes (crises), with uremia, etc.

For the microscopy of the stools, Plate 11, Fig. 2, should be consulted.

The most important parasites found in the stools are cholera-bacilli, typhoid-bacilli, tubercle-bacilli, amebæ, and certain worms. (See page 120 *et seq.*) When helminthiasis exists microscopic examination discloses the presence of large numbers of the respective ova, and at times also Charcot-Leyden crystals (see Plate 8, Fig. 5).

IV. EXAMINATION OF THE UROPOIETIC SYSTEM.

1. Kidneys.

The kidneys extend from the twelfth dorsal to the upper border of the third lumbar vertebra. The right kidney is in contact above with the liver, the left with the spleen.

The kidney becomes palpable only when displaced (wandering kidney), and in the presence of neoplasms: congenital sarcomata, carcinomata (solid tumors, rapid growth, cachexia, hematuria, vermicular blood-clots); when the seat of an echinococcus-cyst (fluctuation, with echinococcus-hooklets, shreds of membrane, and considerable sodium chlorid in the fluid removed by puncture —see Plate 21, Figs. 7 and 8), or of hydronephrosis (alternate filling and evacuation of the cystically dilated pelvis of the kidney, with possible demonstration of urea). These tumors are immovable with respiration.

Percussion.—The normal area of renal percussion-dulness is continuous with that of the liver and spleen respectively, forming with the latter an angle open outward, and extending about 10 cm. (4. in.) laterally from the spinous processes of the vertebra.

Normal renal percussion-dulness is wanting in cases of wandering kidney (more common on the right than on the left). It is increased in conjunction with all forms of tumor of the kidney. As such tumors (in contrast with other forms of tumor) lie necessarily beneath the colon, a tympanitic band appears in the area of dulness on evacuation of the colon, and more distinctly on the introduction of air (see Plate 68).

2. Bladder.

The bladder becomes demonstrable in the median line above the symphysis pubis only when greatly distended. Tumors of the bladder (carcinoma, villous carcinoma) may at times be palpable through the rectum or the vagina. For accurate examination, catheterization and the use of the endoscope are necessary.

3. The Urine.

The urine is secreted from the blood by the kidneys, and contains the excreted salts dissolved in water and the end-products of albuminous disintegration in the body. The results of examination of the urine thus permit of a conclusion with regard to this important metabolic process. They further afford an indication as to the state of the kidneys, in connection with disease of which certain elements appear abnormally in the urine (epithelial cells, tube-casts, albumin, blood), as well as in conjunction with diseases of the remainder of the urinary tract (pelvis of the kidney, ureters bladder, urethra).

Important alterations in the urine occur further in connection with diseases of the heart (cyanotic kidney), with diseases of the liver (biliary coloring-matter), with certain metabolic disorders (diabetes, gout, etc.), with intoxications (lead, iodin, mercury, etc.).

The *amount* of urine secreted in twenty-four hours is 1500 cu. cm. (47 oz.).

Diminution in the amount secreted (under 500 cu. cm.) occurs in conjunction with profuse sweating (fever), diarrhea, cholera (anuria), with diseases of the kidney and of the heart (acute nephritis, cyanotic kidney), with the formation of inflammatory exudations.

Increase in the amount (above 3000 cu. cm., polyuria) occurs after the ingestion of large amounts of fluid, in conjunction with diabetes mellitus and insipidus, with contracted kidney and with the elimination of exudates and transudates.

The *specific gravity* (to be measured with the urometer at room-temperature) is normally 1017 (1010 to 1025). It exceeds 1025 when urine is concentrated and in cases of diabetes mellitus. It falls below 1010 in cases of contracted kidney and of diabetes insipidus.

If the last two figures of the specific gravity be multiplied by 2.33 (Haeser's coefficient), the result represents the proportion of solid elements in grams contained in 1000 cu. cm. of urine.

Color and Sediment.

The normal yellow color of the urine is altered in accordance with the degree of concentration. The more concentrated, the darker; the greater the amount of water present, the lighter. A blood-red color is noted when admixture with blood occurs; a dark-brown color (foam yellow) when jaundice is present (biliary coloring-matter); a dark color (blackish-green) when carbolic acid has been absorbed and when melanin is present (melanosarcoma).

Urine that is clear when first evacuated often becomes turbid on cooling, in consequence of precipitation of uric acid (the precipitate is redissolved upon the application of heat). If the urine is permitted to stand in a conical vessel (sediment), there forms in the lowest portion the so-called nubecula (mucoid cloud). After the urine has stood for some time it becomes turbid in consequence of ammoniacal fermentation (bacterial activity) and there is thrown down a sediment of phosphates, which are redissolved upon the addition of acids. (With regard to the remaining urinary sediments, see Plates 13, 14, 15).

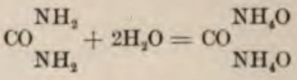
The *reaction* of urine is acid (blue litmus paper is reddened), in consequence of the presence of acid salts (acid sodium phosphate and uric acid).

When the urine is feebly acid it becomes turbid when heated (dissipation of carbon dioxid, and as a result throwing out of solution of earthy phosphates); on the addi-

= – acidity.

tion of acid these salts are redissolved (in contrast with the cloudiness due to albumin).

The urine may be alkaline from the presence of potassium carbonate and sodium carbonate (fixed alkali; red litmus-paper is changed to blue when immersed) through the intermediation of the food; or from the presence of ammonium carbonate (volatile alkali; moistened litmus-paper held above is changed to blue; ammonium chlorid vapor developed when a glass rod moistened with hydrochloric acid is held above); or from the transformation of uric acid into ammonium carbonate (bacterial activity):



$$CO\begin{matrix}NH_2\\NH_2\end{matrix} + 2H_2O = CO\begin{matrix}NH_4O\\NH_4O\end{matrix}$$

The degree of acidity of the urine is greater after the ingestion of meat, cheese, and cereals (from the development of phosphoric acid and sulphuric acid out of albuminous bodies). It is diminished after the ingestion of berries, fruits, and potatoes (potassium carbonate), after the direct administration of alkalies (carbonated waters, etc.), in conjunction with loss of hydrochloric acid (vomiting, hyperacidity), and with chlorosis. = alk

Chemic Examination of the Urine. neutralizing completely

(*a*) *Normal inorganic constituents.*

Chlorids (from 10 to 15 grams—150 to 225 grains—of sodium chlorid daily). The chlorids in the urine are diminished in febrile states and in conjunction with inanition. On the addition of a 10 per cent. solution of silver nitrate to the urine acidulated with nitric acid silver chlorid is normally precipitated in dense flakes; while in pneumonia, for instance, only a slight turbidity results. The quantitative estimation of the chlorids may be made by titration after the methods of Mohr and of Volhard-Salkowski (see text-books of physiologic chemistry).

(1) *Sulphates* (2 grams—30 grains—of sulphuric acid daily). Sulphuric acid appears in the urine united in part with

(1) Rotten eggs SH_2

① [illegible] bodies are from volatile oils.

sodium, potassium, magnesium, and calcium (sulphates), and in part with organic substances, such as phenol, indoxyl, scatoxyl, in the form of ether-sulphuric acids. Through the combination of these aromatic substances, ① resulting from intestinal putrefaction, with sulphuric acid these poisonous bodies are eliminated (thus in cases of carbolic-acid poisoning the sulphates are greatly diminished). The sulphates are precipitated from urine acidulated with acetic acid in the form of barium sulphate upon the addition of a 10 per cent. solution of barium chlorid. The ether-sulphuric acids present in the filtrate are decomposed by boiling with concentrated hydrochloric acid, and are precipitated as barium salts. Their amount affords a means of estimating the intensity of intestinal putrefaction.

Carbonates.—Calcium carbonate, magnesium carbonate, ammonium carbonate. On addition of acids carbon dioxid is set free, with effervescence.

Phosphates.—Acid, neutral, and basic salts, with sodium, potassium, ammonium, calcium, magnesium. In cases of phosphaturia the daily elimination of phosphoric acid exceeds 3 grams—45 grains (to be determined only by quantitative analysis, according to the method of Neubauer).

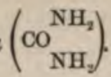
Nitrates and *nitrites* are found in urine in only small amounts.

Sodium (from 4 to 6 grams—60 to 90 grains—Na_2O); *potassium* (from 2 to 3 grams—30 to 45 grains—K_2O); *ammonia* (from 0.5 to 0.8 gram—7½ to 12 grains—daily in fresh urine). The amount of ammonia is increased in cases of diabetes (up to 6 grams—90 grains—daily), in conjunction with diseases of the liver and with fermentation of urine (from urea).

Potassium is present in larger quantity (from three to six times as large) than sodium when there is excessive disintegration of albumin (in cases of fever and of inanition).

Calcium (0.16 gram—2½ grains—CaO); *magnesium* (0.23 gram—3½ grains—MgO daily).

b. Normal Organic Constituents of the Urine.

Urea

$\left(CO\begin{matrix}NH_2\\NH_2\end{matrix}\right)$.

Daily amount from 20 to 40 grams (from 6 to 10 drams); *increased* with excessive disintegration of albumin (diabetes, leukemia, fever, phosphorus-poisoning) up to 100 grams—3 ounces; *diminished* in conjunction with certain diseases of the kidney and of the liver (acute yellow atrophy of the liver), and with inanition.

Qualitative determination (also for the vomited matter in cases of uremia). Evaporation to the consistence of sirup; extraction with alcohol; evaporation of the filtrate; solution in a small amount of water; addition of nitric acid. On cooling, urea nitrate separates in the form of rhombic and six-sided plates.

Quantitative determination of the amount of nitrogen may be made according to the methods of Liebig-Pflüger and of Kjeldahl.

Uric acid ($C_5H_4N_4O_3$); from 0.5 to 1 gram—7½ to 15 grains—daily.

Qualitative Determination by the murexid test.—Evaporation with several drops of nitric acid develops an orange-red spot, which when touched with ammonia assumes a purple color, and becomes blue on addition of potassium hydroxid.

Uric acid is found in urine as acid sodium urate (brick-dust sediment) and as free uric acid; in urine that has undergone decomposition it appears in the form of ammonium urate. It represents the terminal product of disintegration of the nucleins (albuminous substances of the cell-nuclei). It is increased in amount in cases of leukemia (from increased destruction of leukocytes), and at times in cases of gout.

Indican (potassium indoxyl-sulphate) is increased in the urine in the presence of augmented intestinal putrefaction, especially in cases of peritonitis, occlusion of the bowel, perityphlitis, coprostasis, putrid-suppurative processes. It is formed from the indol generated within the intestine, and is demonstrable in the urine as indigo-blue (oxidation).

Test for Indican.—Addition of hydrochloric acid to an equal portion of urine; addition drop by drop of a freshly prepared solution of potassium chlorate, with agitation (not too much to be added); further agitation with chloroform yields a blue color.

The demonstration of indigo-red is without significance (Rosenbach's reaction).

Phenols (phenol hydroquinone, cresol) appear in the urine as ether-sulphuric acids. They also are increased in the presence of intestinal putrefaction.

Test for Phenol.—To 100 cu. cm. of urine are added 5 cu. cm. of concentrated sulphuric acid, and distillation effected. The addition of bromin-water to the distillate yields a yellowish-white precipitate of tribromphenol. Normally from 0.02 to 0.05 gram ($\frac{1}{3}$ to $\frac{3}{4}$ grain) phenol is eliminated daily.

Xanthin and *hypoxanthin* (the latter is abundant in cases of leukemia).

Hippuric acid and *creatinin* have hitherto been considered without significance.

Oxalic acid (from 0.01 to 0.03 gram—$\frac{1}{7}$ to $\frac{1}{2}$ grain—daily) is found especially in the form of calcium oxalate.

c. Pathologic Ingredients of the Urine.

Albumin is generally found in the form of serum-albumin and of serum-globulin, less commonly as albumoses, propeptones, fibrin, and nucleo-albumin. It appears constantly in conjunction with diseases of the kidney (especially nephritis) or of the urinary passages; and transiently in cases of fever, of venous stasis, etc.

It is probable that *physiologic albuminuria* (sometimes cyclic) occurs in healthy individuals, but the amount of albumin eliminated is quite small (less than 0.05 per cent.). The condition has been observed following active exercise, profound excitement, and large meals.

When blood or pus is present in addition to albumin, the latter may have been derived from the plasma of the former (spurious albuminuria). A conclusion as to involvement of the kidneys is to be reached from the presence of other elements (tube-casts, renal epithelium).

The following are the most important tests for albumin:

1. *The Boiling Test.*—The urine is heated in a test-tube to the boiling-point, and several drops of dilute nitric acid are added. The albumin is coagulated, any phosphates precipitated at the same time being redissolved by the acid. The precipitation of the albumin takes place in the test-tube in the course of several hours, and by this means an approximate estimate of the amount may be formed.

Slight turbidity (traces) = 0.01 per cent. albumin.

An appreciable sediment at the bottom of the tube = 0.05 per cent.

The precipitate occupies $\frac{1}{10}$ the column of urine = 0.1 %
" " " $\frac{1}{4}$ " " " = 0.25 %
" " " $\frac{1}{3}$ " " " = 0.5 %
" " " $\frac{1}{2}$ " " " = 1 %

If the entire column has coagulated, the amount of albumin = from 2 to 3 per cent.

2. *Acetic-acid Potassium-ferrocyanid Test.*—The urine is acidulated with several drops of acetic acid, and a 5 per cent. solution of potassium ferrocyanid is added drop by drop. Any albumin present will be coagulated.

3. *Esbach's "quantitative" test* for albumin permits of an approximately quantitative estimation. A graduated test-tube, designated an albuminimeter, is filled with urine to a point marked U, and with Esbach's reagent (consisting of citric acid 5, picronitric acid 2.5, distilled water 245) to a point marked K. The two are well mixed by shaking, and in the course of twelve hours the percentage of albumin can be read from the scale.

Other tests (Heller's nitric-acid test, acetic-acid sodium-chlorid test, etc.) are superfluous. For country practice capsules containing the necessary reagents (citric acid, sodium chlorid, and mercuric chlorid) are serviceable.

Albumoses (formerly taken for peptones) are present in urine especially in conjunction with suppurative processes in some part of the body (pyogenic variety), with ulceration of the bowel, with pneumonia (stage of resolution), with purulent meningitis (in contradistinction from tuberculous meningitis), with a number of diseases of the liver. They may be demonstrated by the biuret-test after removal of albumin by boiling and filtration (development of a violet color on addition of potassium hydroxid and several drops of a 1 per cent. solution of copper sulphate).

Salkowski's Test.—To 20 cu. cm. of urine freed from albumin hydrochloric acid is added, and then phosphormolybdic acid; the resulting precipitate is warmed, washed with water, dissolved in dilute potassium hydroxid, and again warmed until a yellow color appears. On cooling the biuret-test is made.

Nucleo-albumin (formerly taken for mucin). If to the urine, diluted with water and filtered, an abundance of acetic acid be added, nucleo-albumin is precipitated.

Glucose (grape-sugar). Urine containing sugar has a high specific gravity (above 1025). The presence at times of sugar in normal urine has not yet been conclusively demonstrated, although it is probable. Reducing-substances that are not sugar are often present.

Glycosuria is the main symptom of diabetes mellitus. A distinction is made between a mild form (elimination of sugar only after ingestion of carbohydrates (alimentary glycosuria) and a severe form (elimination of sugar also upon a diet of meats and fats; as much as 10 per cent. of sugar may be present).[1]

The most important qualitative tests for sugar:

1. *Reduction-tests.*—Grape-sugar has the property of

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[1] A diabetic patient under my observation excreted on one occasion 1.09 kg. of grape-sugar in twenty-four hours in 9000 cu. cm. of urine.

reducing substances susceptible of reduction (*e. g.*, copper oxid, bismuth oxid). As, however, other substances in the urine (*e. g.*, creatinin, uric acid, turpentine, copaiba, salicylic acid) possess a similar property, these tests are not absolutely conclusive.

Trommer's Test.—To the urine is added one-third its volume of potassium hydroxid (turbidity results from the precipitation of phosphates); then 10 per cent. solution of copper sulphate is added drop by drop. If the urine contain sugar, the hydrated copper oxid formed will remain in solution (clear, azure-blue color), as it will also in urine containing albumin, ammonia, etc. If the urine contain none of these solvent substances, the addition of the copper sulphate results in the formation of a greenish flocculent precipitate. In the presence of sugar there results upon heating the upper layer, and even before boiling, a cloudy precipitate, at first yellowish, then pronouncedly yellowish-red, which gradually distributes itself downward. The blue hydrated copper oxid is reduced to red cuprous oxid. ($Cu(HO)_2$ is converted into Cu_2O.) The test yields a positive reaction in the presence of more than 0.5 per cent. of sugar.

Nylander's Test.—To the urine is added one-tenth its volume of Nylander's reagent (bismuth subnitrate 2, potassium and sodium tartrate 4, sodium hydroxid 100 cu. cm.). In the presence of sugar the upper layer assumes a black color on the application of heat, from precipitation of reduced metallic bismuth. This test will yield a positive reaction when sugar is present in amount equal to 0.1 per cent. and less.

2. *Moore's Test.*—On adding to urine one-third its volume of potassic hydroxid and boiling from two to five minutes a chestnut-brown color is developed, from conversion of sugar into caramel, which may be recognized by its odor. The reaction becomes distinct when not less than 0.5 per cent. of sugar is present.

3. *The Fermentation-test.*—This is the most reliable test for sugar, and is to be employed in all doubtful cases.

Grape-sugar is broken up by fermentation into alcohol and carbon dioxid on addition of yeast. The accumulation of carbon dioxid is the greater the larger the amount of sugar present.

Of three fermentation-tubes, there is introduced into each respectively—

a. The urine to be examined, together with yeast;
b. A solution of grape-sugar, together with yeast;
c. Urine free from sugar, together with yeast.

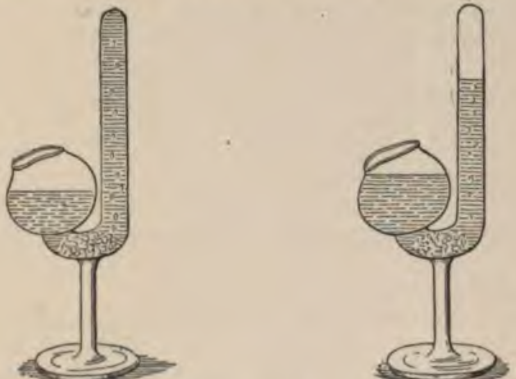


FIG. 31 *a*, FIG. 31 *b*.

Fermentation-tubes (*a*) before and (*b*) after the fermentation of urine containing sugar.

The tubes are permitted to stand in a warm room. After the lapse of twelve hours, often even earlier, carbon dioxid will be present in tube *a* if the urine contain sugar. There should be no carbon dioxid in tube *c*, unless the yeast have contained sugar. Should no carbon dioxid be present in tube *a*, but be present in tube *b*, indicating that the yeast has been active, it may be concluded that the urine is free from sugar.

The test is applicable when not less than 0.1 per cent. of sugar is present.

4. In the *polarization-apparatus* urine containing sugar,

even in as small amount as 0.1 per cent., deflects the plane of polarized light to the right, in greater degree as the amount of sugar present is larger. For this purpose a suitable apparatus is constructed by Schmidt and Haensch, of Berlin.

If the urine be not perfectly clear, it should be filtered after addition of one-tenth volume of lead acetate, and albumin must have been previously removed.

The percentage of sugar contained may be read directly from the scale.

5. *Phenylhydrazin Test.*—A small quantity of phenylhydrazin chlorid (about 0.4) is added to 10 cu. cm. of a warmed 10 per cent. solution of sodium acetate. To the mixture are added 10 cu. cm. of urine, and the whole is placed upon a hot water-bath for an hour. In the presence of sugar there form yellow needle-like crystals of phenylglucosazon, which melt at 205° C. If these occur in small amounts, their presence is to be determined microscopically. Glycuronic acid appears in crystals of similar appearance, but with a lower melting-point.

Quantitative Determination of Sugar.

1. *Fermentation-test* with Einhorn's saccharimeter, which is graduated empirically. Ten cu. cm. of urine are measured and introduced, together with yeast, into a tube. In accordance with its specific gravity the urine must have been previously diluted—if from 1018 to 1022 twice, if from 1022 to 1028 five times, and if from 1028 to 1038 ten times.

More reliable still is the determination with the areosaccharimeter of Schuetz. The flask is filled with urine up to a given mark, and one gram of yeast is added. The vessel is placed in water so that the spindle is immersed, by means of shot, up to the mark 0 per cent. After fermentation for from twenty-four to thirty-six hours at room-temperature the percentage is to be read from a scale.

2. *Titration with Fehling's Solution.*—Fehling's solution consists of copper sulphate 34.639, potassium and sodium tartrate 173, official sodium hydroxid 100, distilled water

sufficient to make one liter. It is best to keep the solutions of the two salts separate, and mix them just before they are to be used.

0.01 gram sugar reduces 2 cu. cm. of this solution.

0.005 gram sugar reduces 1 cu. cm. of this solution.

The urine, especially if it contain a large proportion of sugar, is diluted ten times with distilled water and by means of a buret it is added, drop by drop, to 10 cu. cm. of boiling Fehling's solution, previously diluted with 20 cu. cm. of water, until the blue color of the solution has disappeared. If y cu. cm. of urine have been used, the proportion of sugar is estimated according to the following formula:

$$x = 10. \frac{10 \times 0.005 \times 100}{y} = \frac{50}{y} \text{ per cent.}$$

3. *Polarization Method* (see p. 109).—In addition to grape-sugar, milk-sugar (in nursing-women) and inosite also may be present in the urine, but these are without significance.

Blood.

The urine may present the color of blood—

1. From admixture of red blood-corpuscles (hematuria); and

2. From the presence of free blood coloring-matter in solution, without blood-corpuscles (hemoglobinuria).

The differentiation is to be made by means of microscopic examination.

Hematuria occurs in conjunction with some forms of inflammation of the kidney, with embolism, with nephrolithiasis, with tumors of the kidney and of the bladder, with vesical calculi, etc.

Hemoglobinuria occurs in conjunction with some forms of poisoning, also after transfusion, burns, etc., and together with hemoglobinemia.

On spectroscopic examination there will be found the blood-spectra delineated in Plate 6, and more particularly the spectrum of oxyhemoglobin, of methemoglobin, and of hematin. In addition, the spectrum of hematoporphyrin (two bands in the yellow, etc.) will be found in cases of poisoning with trional and with sulfonal.

Heller's Test for Blood.—The addition of one-third potassium hydroxid destroys the erythrocytes, and the hemoglobin is set free. On boiling, the latter is carried down with the precipitate of phosphates as a brownish-red flocculent sediment.

Biliary Coloring-matter.

When jaundice is present bilirubin (which on oxidation is converted into biliverdin) appears in the urine. On the other hand, hydrobilirubin (urobilin), reduced in the intestine from bilirubin through putrefactive processes, is absent when access of bile to the bowel is entirely cut off. Icteric urine is dark brown in color, and its foam yellow. When shaken with chloroform the bilirubin is dissolved and gives rise to a yellow color. If the urine be carefully poured into a vessel containing concentrated nitric acid, together with several drops of fuming nitric acid, changing rings of color are formed—green, violet, red, yellow (*Gmelin's test*).

To demonstrate the presence of *biliary acids* (in the presence of jaundice) gradual evaporation of a mixture of five drops of urine, a little cane-sugar, and one drop of sulphuric acid in a porcelain dish gives rise to a purple color.

Acetone and diacetic acid are found in the urine when albumin is disintegrated in large amounts (high fever, advanced tuberculosis, diabetes, carcinoma, etc.).

Diacetic acid yields a Bordeaux-red color on addition of iron chlorid. Its presence is indicative of threatened diabetic coma, although it occurs under other conditions as well. At the same time, oxybutyric acid also is often present.

Acetone yields, after over-distillation of 500 cu. cm. of urine acidulated with hydrochloric acid, a yellowish-white precipitate of iodoform on addition of several drops of Lugol's solution of iodin with potassium hydroxid (Lieben's test).

Melanin occurs, also in the form of melanogen, in the urine of patients suffering from melanotic tumors. When the urine is permitted to stand a dark color develops, which on addition of iron chlorid becomes black.

Fat gives rise to a fine milky turbidity (chyluria). It is dissolved upon shaking with ether after addition of potassium hydroxid. Chyluria occurs in association with filaria sanguinis hominis (in the tropics), and with occlusion of the thoracic duct, etc.

The Diazo-reaction of Ehrlich.—A number of febrile diseases (typhoid fever, pneumonia, miliary tuberculosis, septicemia, pulmonary tuberculosis, etc.) are attended with the presence in the urine of certain not intimately known aromatic substances which yield a red color in the presence of sulfodiazobenzol. A mixture of 50 cu. cm. of sulphanilic solution (sulphanilic acid 0.5, hydrochloric acid 5.0, distilled water 100) and 1 cu. cm. of sodium-nitrite solution (sodium nitrite 0.5, distilled water 100) is added to urine in equal parts, together with one-eighth volume of ammonia, and the whole combination is agitated. The foam, as well as the fluid, should be bright red to make a positive reaction. The reaction is characteristic of typhoid fever, and is wanting in cases of meningitis.

Hydrogen sulphid in fresh urine (sometimes attending cystitis) is readily recognized by its offensive odor. A strip of paper impregnated with lead acetate becomes brown on exposure, from the formation of lead sulphid.

Pneumaturia (see p. 125).

Medicaments in the Urine (see Plate 21).

Among the drugs that may appear in the urine the following are worthy of mention (for details textbooks of toxicology should be consulted):

Antifebrin.—On boiling the urine with hydrochloric acid, cooling, and addition of 3 per cent. solution of carbolic acid and iron chlorid a red color is developed that becomes blue on addition of ammonia.

Antipyrin.—Addition of iron chlorid yields a red color.

Lead.—Organic substances present are destroyed by evaporation, with addition of hydrochloric acid and a small amount of potassic chlorate to the point of decolorization. The chlorin is then driven off, and sodium hydroxid added until the reaction is feebly acid. Exposure to hydrogen sulphid yields a brown color due to lead sulphid.

Bromin.—Addition of fuming nitric acid and agitation with chloroform yield a yellowish color.

Carbolic Acid.—The urine exhibits a dark-green color. The test is described on p. 105; the test for ether-sulphuric acid on p. 103.

Balsam of Copaiba and Oil of Sandalwood.—Application of heat and addition of hydrochloric acid yield a pretty red color.

Iodin.—Addition of fuming nitric acid and agitation with chloroform yield a reddish-violet color.

Rhubarb and senna (chrysophanic acid), *santonin* (urine of straw-yellow color).—On addition of sodium hydroxid a red color develops that disappears again when santonin is present.

Salicylic Acid.—Addition of iron chlorid yields a violet color.

Turpentine.—The urine has an odor of violets.

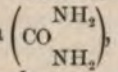
Examination of Renal and Vesical Calculi.—These may be constituted of *uric acid*, and are then hard, smooth, and between yellow and reddish-brown in color. They are recognized by the murexid test (see p. 104). *Calcium-oxalate* calculi resemble mulberries in appearance, and are very hard, nodular, and brownish in color; they dissolve in mineral acids, without effervescence. *Phosphatic* calculi are soft, friable, and whitish in color. Calculi formed of *carbonates* are chalk-like and smooth, and exhibit effervescence on addition of acid. *Cystin* calculi are small, smooth, and yellowish in color; they are soluble in ammonia and on evaporation of the solution they form six-sided crystalline plates recognizable microscopically. *Xanthin* calculi are cinnamon-brown in color, and exhibit a shiny surface when rubbed together.

(For details of analyses larger text-books should be consulted.)

Microscopy of Urinary Sediments.—See Plates 13 to 19.

V. EXAMINATION OF ABNORMALITIES OF METABOLISM.

To maintain the stability and the continuity of the substances constituting the human body (albuminous bodies, fats, carbohydrates, water, and salts), as well as for the generation of energy and heat, the materials burned up in the accomplishment of these purposes (excrementitious products) must be adequately replaced. This is effected by the ingestion of nutritive substances containing the chemic substances named (food-stuffs) in sufficient amount.

1. The *albuminous bodies* are nitrogenous substances of complex constitution which are contained in meat (20 per cent.), milk (4 per cent.), bread (8 per cent.), cheese (30 per cent.), legumins (21 per cent.), rice (8 per cent.), vegetables (2 per cent.), eggs (14 per cent.), etc. They are converted in the stomach and intestines into soluble albumoses and peptones, and as such absorbed. Within the body they undergo various syntheses (muscle-albumin, hemoglobin, nuclein, and many other substances) and forms of disintegration. They break up, in part during the process of digestion, in part later within the body, into a urea-containing (nitrogenous) and an aromatic remnant (C_6H_5-containing). Both of these are excreted in the urine, the one as urea  $\left(\mathrm{CO}\begin{matrix}\mathrm{NH_2}\\ \mathrm{NH_2}\end{matrix}\right)$, ammonia, creatin, and hippuric acid, and the other as phenol, indol, skatol, combined with sulphuric acid (see p. 103). Uric acid is derived from the disintegrated nuclein. A further remnant of the albuminous bodies must be a fat-like body, inasmuch as fat may be accumulated upon an exclusively albuminous diet.

The albuminous bodies contain 16 per cent. of nitrogen (albumin : nitrogen = 6.25 : 1). A certain proportion of these is absolutely necessary for the maintenance of the bodily nutrition (from 90 or 100 to 120 grams daily—that is, from 14 to 20 grams nitrogen). If less be given, the body burns up its own albumin, and suffers a corresponding loss. If more albumin be given with the food, more also is destroyed and more nitrogen is eliminated in the urine, so that the body is thus maintained in a condition of nitrogenous equilibrium.

In the production of energy and of heat the metabolism of albumin is not increased, the body eliminating, whether at rest or engaged in activity, other things being equal, the same amount of nitrogen. On the other hand, a debilitated individual destroys comparatively less daily than a robust person supplied with an abundance of albumin; so that the latter has need also for a larger supply of albumin.

Simultaneous administration of carbohydrates and fats leads to diminished destruction of albumin, so that a certain portion of the albumin-need may be supplied through these articles of food.

Abnormal increase in the amount of albumin-destruction in the body (therefore also in the nitrogenous elimination) takes place in febrile states and in cachectic diseases (carcinoma, profound anemia, leukemia, chronic nephritis).

The measure of albuminous metabolism is furnished by the determination for twenty-four hours of the amount of nitrogen in the food, in the urine, and in the stools. Nitrogenous metabolism = nitrogen present in the urine + nitrogen present in the stools − nitrogen ingested with food. The stools contain about 0.8 of nitrogen, in the fasting state 0.2 per day, as determined by the method of Kjeldahl, which is described in works on physiologic chemistry.

One gram of nitrogen = 6.25 grams of albumin = 29.4 grams of muscle = 2.143 grams of urea.

When intestinal putrefaction is present in conjunction with inflammatory process, etc., a portion of the albuminous matters is converted by the action of microörganisms into poisonous substances (toxins), the intimate nature of which is as yet imperfectly known. Among these are methylamin, dimethylamin, trimethylamin, cholin, neurin, muscarin, cadaverin, etc.

If the products of nitrogenous metabolism (urea, etc.) are not adequately eliminated in consequence of disease of the kidneys, such as nephritis, there results a condition of intoxication by reason of their retention, such as uremia.

Other substances representing end-products of albuminous disintegration, such as the amido-acids, leucin, tyrosin, and asparagin acid (earlier stages of urea), also appear in the urine in conjunction with some diseases of the liver, especially acute yellow atrophy, the liver constituting one of the most important depots for the formation of urea in the body.

2. *Carbohydrates and fats* (non-albuminous foods) constitute, through their disintegration in the body (oxidation), the main source of energy and heat. Oxidation of the carbohydrates takes place especially in the muscles (glycogen), while the fats, by reason of their large carbon-content, are the main source of heat.

The end-products of the oxidation-processes, especially carbon dioxid, are eliminated from the body through the respiratory process.

The disintegration of non-nitrogenous substances, and likewise the need for these substances, are thus the greater the more energy and heat demanded of the organism. An increase of this metabolism occurs also in febrile states, together with increased heat-production.

In diabetes mellitus the body has in varying degree lost its capability of consuming the carbohydrates. As a result, grape-sugar passes from the blood into the urine. The nutritive needs of the body must therefore be satisfied with albuminous and fatty substances. The disease appears in a severe and a mild form (see p. 107). In severe cases sugar is also formed from albuminous bodies.

Constitution of the Food.

If the body receives the required amount of albumin, the three varieties of nutritive material may be represented, with regard to the further composition of the daily amount of food, in accordance with their metabolic value. As a standard of measurement the heat-unit (calory) is employed—that is, the amount of heat required to raise the temperature of 1 kilogram of water 1° C.

One gram of albumin will furnish 4.1 calories; 1 gram of fat, 9.3 calories; 1 gram of carbohydrates, 4.1 calories. Accordingly, 100 grams of fat are isodynamic with 232 grams of starch, with 256 grams of glucose, and with 211 grams of albumin.

A strong, active individual requires daily at least 3000 calories, increasing to 6000 calories, which are provided for as follows:

Albumin,	120 grams	=	480	calories.
Fats,	60 "	=	540	"
Carbohydrates,	500 "	=	3000	"

Considerable variations from these figures are possible. In judging of the nutritive economy it is necessary always to consider the state of previous nutrition, the expenditure of energy and of heat, the bodily weight, the state of the nutrition, the digestive capability, and the pecuniary cost of the respective articles of food.

Emaciated subjects require only from 800 to 1200 calories, while a healthy man in a state of hunger generates 2300 calories. The guide as to the nutrition is furnished by the bodily weight, apart from the formation and absorption of edema, of exudates, and of hydremia, etc.

Table showing Composition, Food-value, and Approximate Cost of some Articles of Food.

Articles of Food.	Percentage-composition.				Percentage-loss through Stools.			Pecuniary Value in Cents.
	Water.	Albumin and Nitrogen, %.	Fat.	Carbo-hydrates.	Albumin (N. %).	Fat.	Carbo-hydrates.	
Uncooked beef, moderately fat	73	20.91 (3.5)	5.19	0.43				3
Boiled beef	76	21.8 (3.5)	0.9–26		2.65	19.2		3
Roast veal	71	15.3 (3.4)	5.2					3
Hen's eggs (1 = 45 grams)	74	14.1 (2.19)	10.9		2.9	5.		3
Cow's milk	87	4.12 (0.64)	3.9	4.2	8.9	5.7		1
Butter	12	0.5 (0.08)	87.	0.5				5
Cheese	34	32.2 (4.7)	26.6	2.97				3–4
Pork	4		95.6		12.1	17.4	1.6	6
Wheat bread	28	9.6 (1.5)	1.	60.	20.7		1.1	2
Rye bread	37	8.5 (1.3)	0.5	52.5	32.		10.9	1
Farinaceous foods	56	8.7	15.	28.9	20.5		1.6	5
Potatoes	74	2.3 (0.37)		23.3	32.2		7.6	1
Peas (uncooked)	14	21.25 (3.4)	1.17	61.8	22.6		5.3	1
Rice	14	8.31 (1.3)		89.2	20.4		0.9	1
Vegetables	73	2.2 (0.35)	3.9	18.1	18.5	6.1	15.4	1
Apples	86	0.4		13.	Alcohol Constituent.			1
Soup	92	1.1	1.5	5.7				1
Beer	90.7	0.44 (0.08)		5.1	3.6 (3.2–4)			1
White wine	91.3			1.4	7.07			3–5
Red wine	89.6			1.4	8.1			3–6
Sherry	80	0.2		3.27	15.			9–13
Malaga					16.			10–15
Champagne					12.			10–20
Gin					45.			4–8
Cognac					60.			8–13

VI. THE MOST IMPORTANT PARASITES.

The human body harbors constantly upon its surface, within its cavities, and in the intestinal canal a large variety of animal and vegetable parasites, which are in part entirely harmless, and in part more or less injurious.

Parasites that gain entrance into the tissues of the body, into the blood, etc., are always pathogenic. The portal of entry is, as a rule, some lesion of the protecting epithelium, however slight.

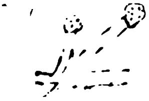
The entrance of parasites may take place through wounds of the skin, of the mouth, of the intestinal tract, through the urethra and the vagina, through inspiration from nose, trachea, or lungs. The injurious effect may be brought about by a direct influence of the parasites upon the nutritive condition of their host, as by the removal of blood or of nutritive substances, or by the formation of certain metabolic products, as ptomains, toxins, toxalbumins, which give rise to poisoning or intoxication. These substances may exert only a local effect, as shown by inflammation, suppuration, or necrosis, or a general effect, as manifested by fever, nutritive disturbances, prostration, coma, and delirium.

a. Vegetable Parasites.

1. **Molds.**—The molds belong to the non-chlorophyllous cryptogams. They consist of a mycelial network, made up of variously branched filaments, upon which spores (conidia) develop. Molds are best cultivated upon sterilized bread-mush. The following varieties are worthy of mention :

a. Mucor corymbifer possesses undivided fruit-bearing organs, at whose extremities the spores develop from the sporangium. In culture it forms a dense snow-white film. It has been found in ulcerated portions of lung and intestine as a secondary infection, and also in the nasal cavity, in the auditory canal, etc. (See Plate 7, Fig. 4.) c. Summer Diarrhoea.

b. Penicillium glaucum possesses branched fruit-bearers.



It develops a (Glaucum) green film upon bread-mush. It is found occasionally in nasal mucus, in sputum, etc., and is without significance.

c. *Aspergillus fumigatus* possesses bulbous fruit-bearers, with free spore-formation. It forms a dirty greenish deposit in cultures. It has been found as a saprophyte in tuberculous caverns, in broken-down carcinomatous nodules, and in abscess-cavities in the lungs (pneumonomycosis aspergillina). Bulb

2. **Budding Fungi, Yeast-fungi.**—These form no mycelium and multiply by budding. They induce alcoholic fermentation in fluids containing sugar. They occur frequently in the gastric and the intestinal contents (see Plate 11, Figs. 1 and 2).

Oïdium lactis is found constantly in milk that is turning sour, etc.

Oïdium albicans, thrush-fungus, belongs to an intermediate variety between ~~molds~~ and budding fungi (see Plate 7, Fig. 2). It occurs in the mouth, the esophagus, and the stomach of children and of debilitated adults (tuberculosis, typhoid fever). It has also been observed in abscesses of the brain.

Certain diseases of the skin are attended with the presence of organisms that belong to the group under consideration:

Favus of the scalp and of the nails is attended with the presence of *achorion schoenleinii;*

Herpes tonsurans of the face, etc., with that of *trichophyton tonsurans;* Ring

(liver spots) tinea –

Pityriasis versicolor with that of *microsporon furfur.*

These are best looked for by teasing in glycerin the structures in which it is suspected that they are contained.

In the mouth and in the deposit upon the teeth may be found constantly *leptothrix buccalis* (see Plate 7, Fig. 1), which probably is an alga. Its filaments are stained blue on addition of Lugol's solution, from the presence of starch-like granules in their interior. The leptothrix contributes to decalcification of the teeth, with the development of caries. It has also been found in the sputum in cases of pulmonary gangrene, and in the pus from cases of abscess of the tonsil.

3. **Bacteria** (for staining and culture, see p. 24 *et seq.*).—These are the lowest form of organisms, multiplying by division (fission-fungi). Besides, under certain circumstances they form, in part at least, also spores. The latter are far more resistant than the mature forms, being destroyed by live steam in not less than three or four hours and by dry heat at a temperature of not less than 140° to 150° C., while the mature forms are destroyed at temperatures over 50°.

a. Micrococci (spherical bacteria).

Staphylococcus Pyogenes (*aureus*, *albus*, and *citreus*, in accordance with the color of the colonies).—The individual cocci are arranged in grape-like masses. The microörganism is the exciting agent causing the more local inflammatory and suppurative processes such as furuncle, abscess, phlegmon, glandular suppuration, otitis media, pyemia, osteomyelitis, and empyema. It has also been found in the blood, in the sweat, and in the urine in conjunction with infectious processes, and also in cases of rheumatic endocarditis, etc. (see Plate 5, Fig. 2; Plate 10, Fig. 2; and Plate 22, Fig. 1).

Streptococcus Pyogenes.—The individual cocci are collected in a chain-like arrangement. The organism attends especially suppuration and inflammation of progressive and ~~malignant~~ character, such as erysipelas, pneumonia, sepsis, especially puerperal, fatal cholera morbus, and as a secondary infection in cases of diphtheria, scarlet fever, etc. (see Plate 22, Fig. 2).

Diplococcus of Fränkel (pneumococcus).—(1) The organisms are arranged in pairs, are biscuit-shaped, and are surrounded with a mucous capsule. They are found abundantly in the sputum in many cases of croupous pneumonia; also in cases of pleurisy, of empyema, of otitis, of endocarditis, of meningitis. They occur also in the

(1) [illegible] like 2 cars side of each other.
[illegible] middle of other (1)

① But never in nucleus.

sputum of healthy persons. Inoculated upon guinea-pigs they induce fatal septicemia (see p. 28; Plate 5, Fig. 3, and Plate 10, Fig. 1).

All of the organisms that have been thus far described stain readily with all of the basic aniline dyes, and remain stained when treated by the method of Gram (see p. 25).

always anywhere *Gonococcus* (Neisser).—This is a biscuit-shaped diplococcus frequently enclosed within leukocytes①. It is found in cases of acute and also of chronic gonorrhea, in cases of ophthalmia of the newborn, of specific cystitis, of endometritis, of salpingitis, and, rarely, in cases of gonorrheal endocarditis, gonorrheal gonitis, etc. The organism can be stained with a mixture of methylene-blue and fuchsin or with gentian-violet (see Plate 19, Fig. 5).

Micrococcus tetragenus.—This organism occurs in packets constituted of four individual cocci. It is found frequently in the sputum, especially in cases of tuberculosis, in addition to tubercle-bacilli (cavity-formation). Rarely

Sarcina ventriculi.—This organism is found in packets constituted of four, eight, sixteen, or thirty-two cocci (see Plate 11, Fig. 1). It is found with especial frequency in the presence of stagnation of the gastric contents and abnormal fermentative processes, such as attend pyloric stenosis and dilatation of the stomach.

b. Bacilli (rod-shaped bacteria).

Anthrax-bacillus.—This organism appears in the form of thick, plump rods, which are found in the anthrax-pustule and in the blood and pus in cases of pulmonary, intestinal, and splenic anthrax. (Plate 5, Fig. 1; culture, see p. 26.)

Tubercle-bacilli.—These are slender, slightly curved rods, which are found in the sputum of cases of pulmonary tuberculosis, in the urine in cases of vesical or renal tuberculosis, in the pus in cases of tuberculosis of bone and in cases of empyema, in the blood in cases of miliary tuberculosis, in the feces in cases of intestinal tuberculosis and also when the sputum is swallowed, in the skin in cases of lupus and of glandular abscesses.

The smegma sometimes contains similar bacilli, but these are decolorized in absolute alcohol within one minute, in contrast with tubercle-bacilli (Plate 10, Figs. 3 and 4; Plate 19, Fig. 6. For specific stain, see p. 25. For inoculation, see p. 28.).

Leprosy-bacilli.—These resemble tubercle-bacilli, but are somewhat shorter. They are found in cases of leprosy of the skin, and of the mucous membranes, and of the nerves. Inoculation is usually attended with negative results.

Typhoid-bacilli.—These are short bacilli, with rounded flagel extremities. They are found in typhoid ulcers, the mesenteric glands and the spleen in cases of typhoid fever; rarely in the suppurative processes that follow typhoid fever. They are demonstrable in the stools only by culture (Plate 22, Fig. 6. For culture, see p. 27.).

Glanders-bacilli.—These are small rods resembling tubercle-bacilli, often exhibiting bright spots. They are found in the pus from glanders-nodules that appear in hostlers, etc. (Plate 22, Fig. 5.)

Tetanus-bacilli.—These are long, slender rods, with spore-formation at one extremity, giving rise to a nail-like or drum-stick shape. They are found especially in earth. Contamination of wounds on the feet or the hands leads to the formation of tetanus-toxin and its absorption, with the development of tetanus.

Diphtheria-bacilli.—These are short, slender rods often club-shaped. They cause diphtheric inflammatory processes and their sequelæ, such as neuritic paralysis, in consequence of intoxication resulting from the development of poisons. Frequently, mixed infections take place from secondary deposition of streptococci, etc. (For culture and staining, see p. 27, Plate 7, Figs. 5 and 6.)

Influenza-bacilli.—These are slender rods, often arranged in pairs, that are found in the sputum and nasal mucus in cases of influenza, and of influenza-pneumonia, etc.

Cholera-bacilli.—These are small curved, comma-shaped rods that are found in the dejecta and in the intestinal contents of cholera-patients. (For culture, see p. 27.)

Bacterium coli commune.—This is an oval rod closely resembling the typhoid-bacillus. It occurs regularly in the intestinal contents, and is not rarely the exciting agent, causing suppurative processes in the abdomen, the kidney, the liver, and also peritonitis, cystitis, pyelitis. (For the differential diagnosis from typhoid-bacilli, see p. 27, Plate 22, Fig. 3.)

Bacterium lactis aërogenes.—This organism resembles the bacterium coli. It occurs in the stools of nursing infants and also in those of adults. Introduced into the bladder through catheterization, it may bring about fermentation and pneumaturia.

c. *Spirilla* (spiral bacteria).

Spirilla of Relapsing Fever (Obermeier).—These are extremely delicate, corkscrew-like organisms exhibiting active motility. They are found during the attack in the blood of patients suffering from relapsing fever (Plate 5, Fig. 4). Cultivation has thus far not been successful.

Spirochæta buccalis.—This organism resembles the spirilla just described, and is found in deposits upon the teeth. It was present in enormous numbers in a case of severe stomatitis under immediate observation (see Plate 7, Fig. 3).

Actinomyces-fungus.—This organism occupies a peculiar position. It belongs actually among the filamentous fungi, forming a dense mycelium, whose periphery is formed of swollen, club-shaped structures representing degenerated involution-forms. Macroscopically it forms sulphur-yellow granules, each of the size of a pin's head. It is the cause of a number of chronic destructive infiltrating and suppurative processes in the lungs and of bones and joints, especially in the submaxillary region. It is conveyed to man by cattle (see Plate 10, Fig. 6).

(For fuller details concerning the foregoing parasites reference should be made to the larger works on bacteriology.)

b. Animal Parasites.

1. Insects.

Several varieties of insects occur as ectoparasites upon the skin of man: *pediculus capitis* and *vestimenti* (the head-louse and the body-louse); *pulex irritans* (the human flea); *acanthia lectularia* (the bed-bug). *Pulex penetrans* (the penetrating flea, found in South America) and *ixodes ricinus* (the common tick) are bloodsuckers, as is also *argas reflexus* (the pigeon-tick), which is only occasionally encountered in man. Further, there are *phthirius pubes* (the crab-louse), found on the mons veneris; *demodex folliculorum* (found in the hair-bulbs in the presence of comedones); *sarcoptes scabiei* (the itch-parasite, which bores large passages beneath the horny layer of the epidermis, in which it deposits its ova).

2. Vermes.

These occur especially in the intestinal canal as parasites. Often without significance, they may occasionally, by their presence in large numbers or by gaining entrance into the ducts of glands, give rise to profound disturbances.

a. Round-worms (nematodes).

Ascaris lumbricoides (spool-worm).—Infection occurs through the introduction of ova by way of the mouth from without (see Plate 11, Fig. 2). Development takes place in the small intestine in children and in the mentally deficient. The males are half as large as the females.

Trichocephalus dispar (Fig. 32, *a*, male; *b*, female).—This parasite is found in the cecum. With the aid of its whip-like cephalic extremity it bores its way superficially into the mucosa.

Oxyuris vermicularis (Fig. 32, *c*, male; *d*, female. Ova: see Plate 11, Fig. 2).—This worm is found in the colon, the impregnated female in the cecum. Autoinfection occurs through scratching in the anal region in consequence of the itching.

Ankylostomum duodenale (Fig. 32, *e*, *f*. Ova: Plate 11, Fig. 2).—This parasite is found in the upper portion of the small intestine, where, by means of strong mouth-hooklets, it bores its way into the mucous membrane and sucks the blood of its host. It is capable of producing a high degree of anemia. It has been found in the tropics, in Italy, among workmen in the St. Gotthard tunnel, in brick-burners.

Anguillula Intestinalis.—This worm is found in the small intestine, often in association with the ankylostomum. It appears to be without significance.

Trichina Spiralis (Fig. 33).—The muscle-trichina gains entrance into the stomach and intestine of human beings with trichinous pork. When its capsule is dissolved the intestinal trichina is set free and undergoes fructification. The young embryos bore through the intestinal wall, gain entrance into the blood, and finally become encapsulated in the muscles. Here they remain capable of development for years, especially in the diaphragm, the abdominal muscles, those of the larynx, the facial muscles, and the ocular muscles.

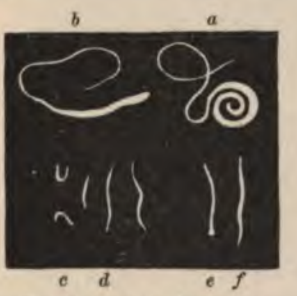


FIG. 32.—Intestinal parasites.

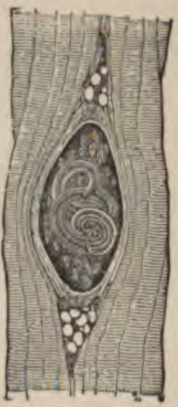


FIG. 33.—Recent muscle-trichina (after Heller).

Filaria sanguinis and *filaria medinensis* occur in the tropics. The first, through the circulation in the blood of its embryos, causes hematuria and chyluria. It may also be eliminated with the urine. The second induces furunculosis.

b. Cestodes (tapeworms).

These gain entrance to the human intestine as cysticerci contained in the flesh of an intermediate host, and they develop into true tapeworms, provided with a head or scolex and segments or proglottides.

Tænia Saginata.—This worm is from 4 to 5 meters long. Its head is provided with four suckers, without hooklets. The uterus is markedly branched (see Fig. 34, *a*. Ova: see Plate 11, Fig. 2). This parasite is conveyed through beef.

Tænia Solium.—This worm is from 2 to 3 meters long. Its head is provided with a crown of hooklets and with four suckers. The uterus is less markedly branched (see Fig. 34, *b*. Ova: see Plate 11, Fig. 2). Conveyance takes place through pork. The larval form (cysticercus cellulosæ) may also gain access to man and occasion development of cysticercus in brain, skin, eye, muscles (see Fig. 35).

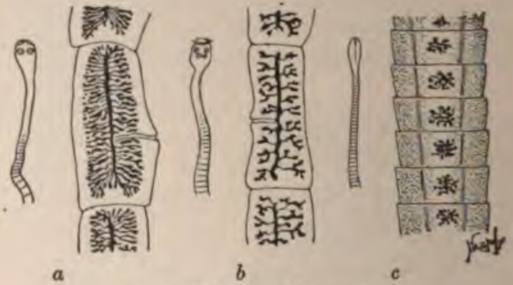


FIG. 34.—Varieties of tapeworm.

Tænia Nana.—This worm occurs only in southern countries—*e. g.*, Italy and Egypt.

Tænia Echinococcus.—This occurs in the larval state in the human being (the ova being derived from the intestine of the dog) in liver, kidney, lung, heart, where it forms large cysts in whose interior hooklets, portions of membrane, etc., are found (see Plate 21, Figs. 7 and 8).

Bothriocephalus latus (see Fig. 34. Ova: see Plate 11, Fig. 2).—This worm is from 4 to 10 meters long. The intermediate host is probably some variety of fish. The parasite is a frequent cause of profound anemia.

c. *Trematodes* (*sucking-worms*).

Distoma hepaticum is sometimes found in the biliary passages of human beings.

Distoma hæmatobium is found in the tropics in the veins of bladder and rectum and gives rise to hematuria, ulceration, and diarrhea.

Distoma pulmonale is found in the upper air-passages and gives rise to hemoptysis. The sputum may contain ova, together with Charcot-Leyden crystals.

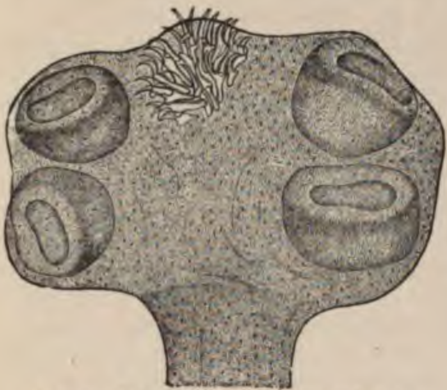


FIG. 35.—Head of a cysticercus (after Heller).

3. **Protozoa.**

Various infusoria (flagellates, ciliates) belong to this group. Cercomonas, trichomonas (see Plate 11, Fig. 2), are found in the intestine, in the feces, in the vagina, in abscesses. Similar forms are said to have been found in the blood in cases of pernicious anemia.

Megastoma entericum occurs in diarrheal and in normal stools, often encysted.

Amœba coli is found in the large intestine, and in lar number in fecal abscesses and in some cases of dysente A causative connection is probable.

Gregarines are found at times in the intestinal conten in the liver, and in tumors.

Plasmodia are found in the blood in the various for of malaria (intermittent fever). They are to be look upon as the cause of these varieties of fever, inasmuch they gain entrance into the red blood-corpuscles and destr them. They thus ~~transform~~ the contained hemoglob into ~~black pigment~~. Some of the organisms are ciliate some crescentic. The more common forms are depict in Plate 5, Figs. 5 and 6.

The plasmodia are found in largest numbers short before the paroxysm, and during the rise of temperatu The majority disappear during the decline of temperatu and only small bodies remain visible. Propagation tak place through spore-formation. The course of the spor lation (segmentation) is believed to determine the type the fever (tertian or quartan). In the quotidian type is thought that two generations are present which u dergo maturation on successive days. The ameba tertian fever divides into fifteen or twenty spores; th of quartan fever into only six or ten (Golgi).

Blood-preparations, after fixation, may be stained wi methylene-blue and eosin (see p. 78, *et seq.*).

Recently a motile rhizopod, Leydenia gemmipara, h been found in ascitic fluid from a case of carcinosis the peritoneum. Concerning this, however, further i vestigation is necessary.

SECTION IV.

EPITOME OF SPECIAL PATHOLOGY AND TREATMENT.

I. INFECTIOUS DISEASES.

A. Bacterial Infections.

1. *Tuberculous Diseases.*—If *tubercle-bacilli*, derived from the sputum of tuberculous subjects or the secretions of diseased animals, gain entrance into the body through small wounds of the skin (by way of the lymph-channels), through the inspired air, or through ingested food, they excite at the place of localization the formation of small granulation-tumors—tubercles. These consist of an accumulation of leukocytes and hyperplastic fixed cells of the tissues. These tubercle-nodules undergo necrosis (caseation) and disintegration, leaving ulcerous areas that extend and become confluent, so that the affected organ is gradually destroyed. Frequently the tuberculous ulcer becomes the seat of secondary infection (mixed infection) with pyogenic cocci and other microörganisms, which in turn bring about secondary inflammatory processes (infiltrations, septic diseases). The cure of the tuberculous process takes place through absorption, connective-tissue hyperplasia (cicatrization), and calcification.

From German statistics begun in 1892, bearing upon the causes of death, with reference to age, it appears that, among 1000 deaths in the year 1893, between 105 and 107 were due to tuberculosis. If these are classified according to age, it is found that among 1000 deaths in children in the first year of life, only 10.8 are due to tuberculosis;

among children between one and fifteen years of ag
62.2 are due to tuberculosis; among persons betwe
fifteen and sixty years of age, 322.3; and among tho
over sixty years old, only 60. Tuberculosis thus destro
principally persons at the productive period of life, and c
those dying at this time one-third owe their death
tuberculosis.

Localized Tuberculosis.

a. Pulmonary Tuberculosis.

Predisposition to tuberculosis exists in individuals wit
hereditary tendencies (in particular between the fifteent
and thirtieth years), poor nutritive conditions, deformiti
of the chest, congenital lesions of the heart, tuberculo
habitus, etc.

The *first symptoms* are emaciation, anemia, slight febril
symptoms, obstinate cough, hoarseness. As the case pr
gresses, there will be found a catarrhal condition of t
apex of one lung (tuberculous bronchitis), with heigh
ened or enfeebled vesicular breathing, prolonged expir
tion, fine moist râles, impaired resonance over the ap
of one lung, flattening of the same side, retardation o
movement in respiration, due to adhesive pleurisy, wit
blowing, jerky breathing, and râles. The diagnosis
made certain by the demonstration of tubercle-bacilli i
the sputum (see p. 25).

At an *advanced stage* there are progressive emaciatio
abundant expectoration, and cough. The sputum co
tains elastic fibers and tubercle-bacilli. Night-swea
occur and hectic fever is present. There is dulness ov
the entire upper lobe, due to tuberculous infiltration, wit
bronchial breathing and ringing moist râles of modera
size. Hemoptysis (frothy light or dark-red blood accor
ingly as the source is an eroded artery or vein) may tak
place, and there may be dry pleurisy or exudative ple
risy.

In the *terminal stage*, in addition to the foregoing sym
toms, there may be present over the upper lobes a tym
panitic note upon percussion, changing with variations i

posture and with opening and closure of the mouth (see p. 45); ringing moist râles of large size and amphoric

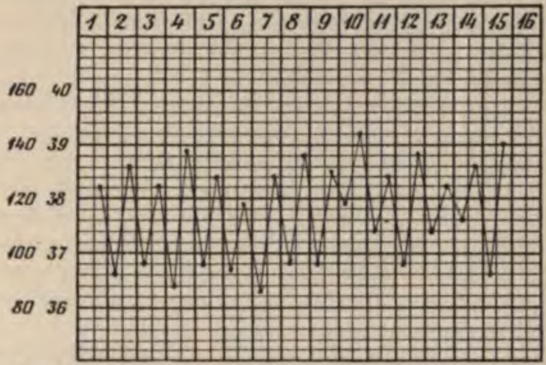


FIG. 36.—Temperature-chart from a case of chronic tuberculosis, with remittent (hectic) fever.

breathing; together with large moist râles also over the lower lobes.

Complications. — Laryngeal tuberculosis (persistent hoarseness); intestinal tuberculosis (diarrhea); pleurisy; pericarditis; contraction of the lung; miliary tuberculosis; hemoptysis; pneumothorax; amyloid degeneration; nephritis; neuritis.

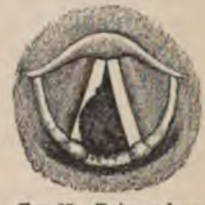


FIG. 37.—Tuberculous laryngitis.

Treatment.—Nutrition (milk, kefyr, whey, eggs, cod-liver oil, butter, extract of malt). Alcohol is superfluous. Cool sponging, much out-of-door life; a southern climate in winter or a high altitude (treatment in a sanatorium): Falkenstein, Görbersdorf, Meran, Reichenhall, Davos, Riviera, and many others. Arsenic, creosote, myrtol, solveol, etc., in pills. Inhalations of ethereal oils (oil of pines). Mineral waters (Soole, Ems, Selters, etc.). Expectorants (pectoral tea, senega, apomorphin, ammonium chlorid, etc.). To control cough: codein, opium, belladonna, morphin. For night-sweats: cool sponging, atropin, agaricin, etc. For hemoptysis: rest, morphin, ice-bag, ergotin, lead acetate, etc. For severe febrile manifestations: antipyretics. Individual sputum-cup. Disinfection of linen, etc.

b. Glandular Tuberculosis.

This occurs frequently in children. When associated with cutaneous eruptions, morbid development of adipose tissue, anemia, diseases of nose and ear (chronic catarrh), it is designated scrofulosis. The glands most commonly affected are those of the neck (and especially the submaxillary glands), the bronchial glands, the mesenteric glands, the axillary glands, etc. The glandular enlargements may continue for a long time, finally softening and undergoing suppuration.

Treatment.—Nutrition, etc. (see Pulmonary Tuberculosis). Cod-liver oil with lime, iron iodid, arsenic. When accessible, surgical extirpation.

c. Laryngeal Tuberculosis.

This is usually secondary to involvement of the lungs through the sputum. Tubercles form upon the vocal bands and undergo ulceration, giving rise to imperfect approximation, to hoarseness, and to toneless cough. The interarytenoid spaces, the ventricular bands, and the epiglottis also may be affected, with resulting pain on swallowing.

Complications.—Edema of the glottis, giving rise to suffocative attacks; laryngeal perichondritis, with abscess-formation.

Treatment.—Inhalations of balsam of Peru, of tannic acid, etc.; cauterization with lactic acid; applications of cocain if there be pain; of morphin solution; gargling with ice-water. Surgical treatment may be indicated if the pulmonary disease be not too far advanced.

d. Intestinal Tuberculosis.

This usually develops secondarily through the agency of swallowed sputum. As a result, ulcers form throughout the entire intestinal tract, particularly in the cecum, and there occurs persistent diarrhea, with bloody and mucous stools and abdominal pain. Perforative peritonitis and intestinal hemorrhage may occur as complications. The discovery of tubercle-bacilli in the feces is not absolutely conclusive, as they may be derived from the sputum.

Treatment.—Opium, poultices, bismuth subgallate, astringents (silver nitrate, tannic acid, etc.), mucilaginous drinks, arrowroot, decoction of salep and colombo.

e. Pleurisy.

Dry pleurisy is frequently a secondary manifestation of pulmonary tuberculosis, and is attended with pleuritic friction and pain in the side. Exudative pleurisy occurs frequently in association with latent or not far-advanced pulmonary and glandular tuberculosis. It is attended with an eruption of tubercles upon the pleura. The exudate is mostly serous, rarely hemorrhagic. It is often sterile, although it may contain tubercle-bacilli, as demonstrated by inoculation.

For other symptoms and treatment, see Pleurisy (p. 172).

Less common is *tuberculous pericarditis*, tubercle-formations taking place upon the layers of the pericardium (see Pericarditis, p. 186).

f. Urogenital Tuberculosis.

Tubercle-bacilli are almost constantly eliminated with the urine whenever tuberculosis is present anywhere in the body, without the urinary passages being necessarily involved. Renal tuberculosis, vesical tuberculosis, etc., may, however, be present at the same time. The most significant manifestation of these disorders is an abundant admixture of pus with the urine. Nephritis alone occurs often in subjects of tuberculosis without being specific (toxic parenchymatous nephritis). Primary tuberculosis of the testicle, the seminal vesicles, the prostate, etc., is sometimes observed.

Treatment.—Eventual surgical extirpation of all diseased parts.

Generalized Tuberculosis.

g. Miliary Tuberculosis.—This results from the rupture of a tuberculous focus (cavern, gland) into the blood-stream, especially a vein. The distribution through the circulation gives rise to extensively disseminated occurrence of tubercles in lungs, pleura, pericardium, liver, spleen, kidneys, peritoneum, meninges, etc.

The most important symptoms of miliary tuberculosis of the lungs are the marked dyspnea and the cyanosis in the presence often of only slight objective manifestations, while impairment of resonance at the apices and the detection of bacilli in the sputum are demonstrative of tuberculosis. In other cases symptoms of profound (typhoid) general infection are the more conspicuous, as manifested by prostration, headache, etc. The fever is irregular, often persistently high, although it may be wanting. The course of the disease is often atypical, and its duration may be from a week or ten days to several weeks. The spleen is likely to be enlarged, and tubercles may be present in the choroid. Tuberculous meningitis indicates its presence by rigidity of the neck, vomiting, and strabismus. The most important point, however, is always the demonstration of tuberculosis in other portions of the body.

h. Tuberculous Peritonitis.

This occurs usually in conjunction with tuberculous pleuritis or glandular or intestinal tuberculosis. The dry form of peritonitis is attended with numerous adhesions and contraction of the omentum; the exudative form, with the presence of nodules in the peritoneum and with serous exudation, often hemorrhagic. There may be, besides, vomiting, fever, abdominal pain, diarrhea, and cachexia. The diagnosis cannot always be made with positiveness, especially when the disease is primary.

Treatment.—In cases of exudative peritonitis celiotomy may be undertaken with the expectation of good results. In other varieties the treatment should be symptomatic.

i. Tuberculous Meningitis (Basilar Meningitis).—This develops as a complication of primary tuberculosis of lungs, lymphatic glands, and bones, and with especial frequency in children. The dissemination of tubercle-bacilli through the vessels of the pia-arachnoid is followed by the formation of miliary tubercles, together with a fibrinous exudate at the base of the brain containing miliary nodules. If extension of the nodules to the brain take place, tumor-like solitary tubercles may form.

The disease begins gradually with malaise, headache, and vomiting. In a short time the symptoms become aggravated, with indications of cortical irritation, delirium, convulsions, trismus, and irregularity of the heart. There may be, further, somnolence, a temperature as high as 39° C. (102.2° F.), irregular fever, rigidity of the neck, and coma. Finally, there may be paralysis of isolated cerebral nerves (oculomotor, facial, abducens), monoplegia, aphasia, and eventually death.

The *diagnosis* must be confirmed by the demonstration of tubercle-bacilli in the sputum, or in the fluid obtained on puncture of the spinal column in the lumbar region, or by the recognition of tubercles in the choroid.

The *treatment* includes rest, cold applications (baths are painful), and ice-bag, bleeding from the mastoid region, calomel (gr. iij, thrice daily), cantharidal blisters at the nape of the neck, mercurial inunctions, antipyretics and narcotics.

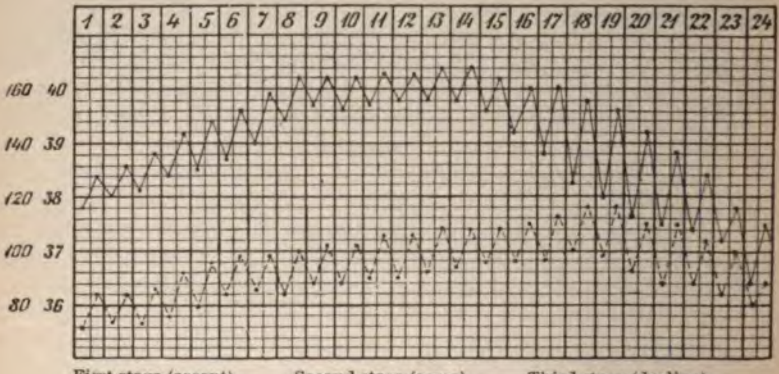
2. *Typhoid Fever.*

Infection with *typhoid-bacilli* (see p. 27) probably takes place usually through the intermediation of the food (milk, water, fruit, etc.), and under circumstances also directly through the dejections, the linen, etc., of typhoid patients. Epidemics result through contamination of water and of soil with typhoid-bacilli.

The bacilli bring about a characteristic morbid process in the small intestine: swelling of the follicles and of Peyer's patches, medullary infiltration, breaking down, and ulceration, with final cicatrization. The spleen and the lymph-glands become enlarged, and in them typhoid-bacilli can be found. Frequently secondary infections are added. The principal symptoms are attributable to the intoxication resulting from absorption of the toxalbumins generated in the intestinal canal.

The period of *incubation* is from ten to twenty days. The *prodromal* symptoms include anorexia, lassitude, headache, and general malaise.

The *initial stage* occupies the first week, and is characterized by fever of slowly ascending type, with severe headache, prostration, constipation, and coated tongue.



First stage (ascent). Second stage (acme). Third stage (decline).

FIG. 38.—Temperature-chart from a case of typhoid fever.

The *second stage* or *acme* of the disease occupies the second week, and is marked by continued fever, with a temperature at about 40° C. (104° F.); a dry, fissured tongue; rose-spots upon the abdomen, thorax, etc.; enlargement of the spleen, recognizable by palpation; gurgling in the ileocecal region; diarrhea with peasoup-like stools; meteorism; diazo-reaction (see Plate 20, Fig. 12). There is no leukocytosis (see Plate 3, Fig. 6). Diffuse bronchitis is usually present. The febrile manifestations are pronounced and the pulse is dicrotic (see Fig. 39).

There may be coma and delirium. The patient occupies the dorsal decubitus, and is exposed to the danger of the formation of bedsores.

The *stage of decline* occupies the third week. The fever begins to decline, falling a degree or more in the morning, and rising slightly less in the evening. Later it continues to subside more gradually.

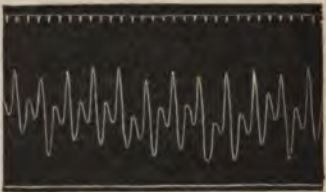


FIG. 39.—Pulse-tracing from a case of typhoid fever, showing dicrotism.

Relapses (see Fig. 40) occur frequently from six to eight days after the decline of the fever, and are of short duration. *Convalescence* may, however, be much retarded in consequence. *Complications* occur most frequently during the last week: intestinal hemorrhage, perforative peritonitis, glandular abscesses, otitis media, parotiditis, venous thrombosis, pneumonia (catarrhal, less commonly fibrinous).

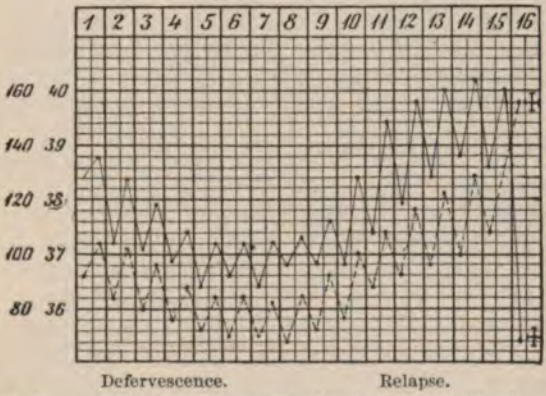


FIG. 40.—Temperature-chart from a case of typhoid fever with relapse, and death from perforative peritonitis.

Diagnosis.—Diagnostic importance is to be attached especially to the characteristic mode of onset (absence of chill, as well as of leukocytosis and of herpes), and the roseola.

The *Gruber-Widal serum-test* yields a positive reaction in almost all cases after the eighth day, and often even earlier. A few drops of blood (about 2 cu. cm.) are obtained by puncture of the finger with suitable aseptic precautions; the serum that separates is introduced into a narrow test-tube containing sterile bouillon, which is then inoculated with living typhoid-bacilli and kept at the temperature of the body. In the course of a few hours agglutination and sedimentation, with loss of motility of the bacilli, take place if the case be one of typhoid fever. A positive reaction is occasionally yielded in cases of other disease than typhoid fever.

The *prognosis* is dependent upon the condition of the heart, the intensity of fever, and possible complications.

Treatment.—Bland, unirritating, liquid diet (milk, broths, eggs, beef-juice, in abundance), calomel, gr. ix, in three doses at the onset. An ice-bag or Leiter's tubes upon the head. Cold packs, cold baths at 20° C. (68° F.), and in case of necessity antipyretics. Later, possibly digitalis and stimulants. In case of hemorrhage, opium, ice-pills, iced milk. To prevent bedsores an air-bed or a water-bed and frictions with alcohol.

3. *Diphtheria and Croup.*

Diphtheria-bacilli (see p. 124) find lodgment most frequently upon the tonsils (pharyngeal diphtheria), the laryngeal, the tracheal, and the bronchial mucous membrane (croup), the nasal mucous membrane (nasal diphtheria); rarely upon the vaginal and the intestinal mucous membrane. Wherever present they give rise to the formation of a fibrinous exudate in the necrotic epithelial layer (diphtheria-membrane). In consequence of the formation of extremely poisonous toxalbumins, as well as the mechanical results due to the presence of the membranous deposits, the most alarming symptoms may result. The young patients are seized with high, irregular fever, often vomiting, pains in the throat, and cough. The symptoms of intoxication are marked acceleration of pulse, coma, vomiting, and collapse. The morbid process may extend from the throat to the nose (septic diphtheria) or to the larynx. Frequently the lymphatic glands of the neck undergo enlargement, with suppuration, due to secondary infection with pyogenic cocci. Suppuration of the middle ear and endocarditis may also occur. Frequently there is hemorrhagic nephritis of toxic origin.

In cases of *croup* there develop soon, in addition to the rough, barking, croupy cough and the hoarseness, the alarming symptoms of laryngeal stenosis. Respiration becomes labored and difficult, inspiration whistling; finally the breathing becomes stertorous, attacks of suffocation occur, and portions of membrane are ejected by cough. Cyanosis may be marked.

Sequelæ: post-diphtheric neuritis (paralysis of accommodation, difficulty in deglutition, abolition of the patellar reflex, ataxia). Chronic nephritis (secondary contracted kidney). Cardiac valvular disease.

Diagnosis.—In cases of angina the diagnosis can only be made with certainty by demonstration of diphtheria-bacilli (see p. 27).

Treatment.—Injection of diphtheric antitoxic serum. Gargles or inhalations of dilute solutions of carbolic acid. Swabbing with solution of carbolic acid. Insufflation of sodium sozoiodol. Sodium iodid. Anisated water of ammonia internally. Stimulants. Cold applications and baths. Iced milk.

Laryngeal stenosis is to be treated with emetics, intubation, or tracheotomy.

4. *Influenza.*

Influenza-bacilli (see Plate 10, Fig. 5) are found in the nasal mucus, the sputum, etc. The severe symptoms of the disease are due especially to the toxins produced. The disease is frequently epidemic in its occurrence. It begins with a considerable degree of fever, great prostration, and pains in head, extremities, and sacrum. Soon are added coryza and cough (catarrhal variety); at times the pains in the extremities are the more conspicuous (rheumatic variety); at still other times the constitutional phenomena are the more pronounced (typhoid variety). The course of the disease is irregular, the duration from three to seven days.

Complications.—Diffuse bronchitis, catarrhal pneumonia, less commonly croupous pneumonia, pleurisy, endocarditis, otitis media, neuralgia, anemia, etc.

The *treatment* is symptomatic, and may require, among other things, the use of antipyretics and of stimulants.

5. *Asiatic Cholera.*

Cholera-bacilli (see Plate 11, Fig. 4) gain entrance into the stomach and intestines, especially in the presence of gastric catarrh with diminished production of hydrochloric acid, with drinking-water, food, etc., from contamination of the soil, through the intermediation of cholera-dejecta, infected linen, etc. In the intestines they form toxalbumins, which give rise to exudation, as well as necrosis of epithelium, and which when absorbed into the circulation bring about toxic manifestations.

The period of *incubation* is several days. After a premonitory diarrhea the cholera attack proper sets in. There occur from ten to thirty ricewater-like stools as a result of intestinal exudation, with vomiting, thirst, collapse. Cramps in the calves occur in the algid stage, with subnormal temperature, hoarseness of voice, cyanosis (inspissation of the blood, weakness of the heart), uremic conditions (anuria, cholera-kidney). Before convalescence sets in the patient may pass through a period of cholera-typhoid, characterized by obscuration of the sensorium, delirium, and insomnia.

Complications.—Heart-failure, hemorrhagic nephritis (uremia), bedsores, pneumonia.

Diagnosis.—Demonstration of bacilli by culture (see p. 27).

Treatment.—Prophylactic isolation of the sick and disinfection of all dejecta. Dilute hydrochloric acid for prophylaxis. Calomel, opium, ice-pills, hot packs, stimulants, infusions of saline solution.

6. *Leprosy.*

Invasion of *leprosy-bacilli* (see p. 124) gives rise to the formation of nodules, with secondary breaking down, in the skin of the trunk, the face, and the extremities (*tuberous leprosy*). There may be also pigmentation of the skin. Leprous extremities may exhibit ulceration, with exfoliation of portions of fingers (*mutilating leprosy*). Leprous neuritis occasions anesthesia and trophic disturbances. The course of the disease is chronic. It occurs in Sweden, Norway, Turkey, and along the East Sea.

7. *Anthrax* (malignant pustule).

Anthrax-bacilli (see p. 123) gain access to man (butchers, brushmakers, ragpickers, etc.) from diseased animals (sheep, oxen and their hides, especially the hair), and multiply in the blood. The portals of entry are the external skin (malignant pustule), the intestine (intestinal anthrax, intestinal mycosis), etc.

Malignant pustule is attended with large carbuncles presenting bluish-black centers, with lymphangitis and lymphadenitis. From these foci general infection may take place, with the development of irregular fever, delirium, diarrhea, collapse.

Intestinal mycosis is characterized by vomiting, diarrhea, fever, cardiac insufficiency, dyspnea, irregularity of the heart, cyanosis, collapse.

Treatment.—Prophylaxis; disinfection and burial at a great depth of all bodies dead of anthrax. In cases of malignant pustule immediate excision should be practised. In other respects the treatment is symptomatic.

8. *Glanders.*

The *bacillus of glanders* (see p. 104) gains access to man (hostlers, drivers, etc.) through the intermediation of horses. It gives rise to the formation of glanders-nodules, which later break down and form abscesses. They occur most commonly in the nose, the bronchi, and the lungs. At the same time the lymphatic glands in the neighborhood of the nodules become enlarged, with subsequent suppuration and the development of erysipelatous and phlegmonous processes. The fever is irregular and often high, and the symptoms of general infection from the generation of toxic products are pronounced.

Treatment.—Prophylaxis; operative removal of infiltrates and abscesses.

B. Coccus-infections.

9. *The Rheumatic Diseases.*

Staphylococci (see p. 122) gain entrance into the tissues of the body through secondary infection in the course of a number of diseases (angina, measles, scarlet fever, influenza, smallpox, typhoid fever, etc.). In accordance with their number and virulence, and in accordance with the varying intensity of reaction of the tissues at the site of their localization (lymphatic glands, middle ear, bone and periosteum, joints, serous cavities [pleura, pericardium], endocardium), they give rise to inflammatory manifestations in the form partly of simple serous exudation and infiltration, partly of cellular infiltration with secondary suppuration.

Similar infiltration may take place also without recognition always of the portal of entry (cryptogenetic variety). Of these diseases, which may be designated as septicopyemic infections in the widest sense, a mild and a severe form may be distinguished, in accordance with considerations already dwelt upon. In the category of the mild form belong acute articular rheumatism, benign endocarditis, rheumatic pleurisy, and pericarditis; while in the category of the severe form belong the pyemic and septicopyemic diseases.

In addition to the form of staphylomycosis just described, streptococci, and with considerably less frequency also, gonococci, further the diplococcus of pneumonia, the typhoid-bacillus, the bacterium coli commune, and others, may in similar manner give rise to secondary infection.

Acute Rheumatic Polyarthritis.—Acute articular rheumatism occurs frequently in typical attacks of from five or seven to eleven days' duration, with intervals of about the same length. The course of the fever is indicated by the temperature-chart shown in Fig. 41.

The disease frequently sets in with slight tonsillitis, intestinal catarrh, etc. (portal of entry for the infection), which may be followed by a chill. The joints and the upper and lower extremities successively undergo swelling, and severe pain is suffered on the slightest movement. Perspiration is profuse, and frequently cutaneous eruptions appear (sudamina, urticaria, erythema). After a period free from febrile manifestations and pain a repetition of the attack occurs. Similar cycles are repeated, in accordance with the severity of the infection, in the course of which the following complications may arise:

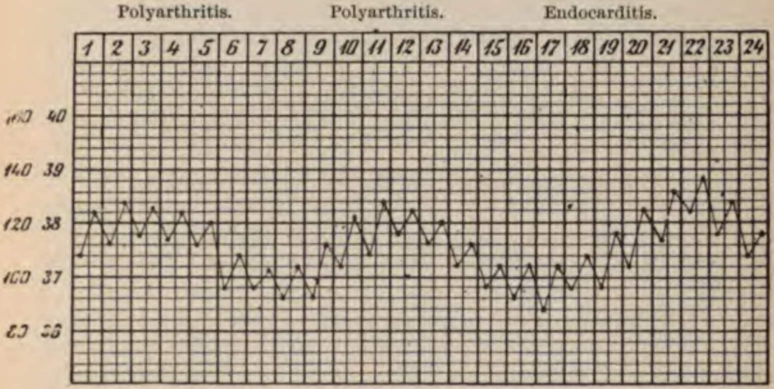


FIG. 41.—Temperature-chart from a case of acute rheumatic polyarthritis.

Acute Endocarditis (see also p. 177).—This is observed in 20 per cent. of all cases. It is characterized by fibrinous inflammation of the endocardium, of the valves of the heart, in consequence of the lodgment of the cocci, with the development of a blowing especially systoïic murmur, dilatation of the heart, and elevation of temperature.

The endocarditis may in turn be followed by valvular lesions (insufficiency or obstruction).

Acute Rheumatic Pleurisy.—Serous effusion into the pleural cavities; also fibrinous and exudative pericarditis (see also p. 172). At times acute articular rheumatism is complicated by the development of chorea minor (St. Vitus' dance), which, it has been thought by some, is due to disseminated areas of encephalitis, and by others to cerebral embolism.

In cases of cerebral rheumatism the temperature may reach an excessive height, up to 43° C. (109.4° F.), and there may be delirium and collapse. In severe cases the muscles of the extremities sometimes remain paralyzed and atrophic for some time. The attack may be followed by the development of subchronic and chronic forms of rheumatism, with thickening of the joints, stiffness, and pain.

Treatment.—Salicylic acid, gr. viiss, hourly; sodium salicylate, gr. xlv—gr. lxxv, twice a day; salophen, gr. xxx, three times daily; salol, gr. xv–xxij, thrice daily; wet packs; enveloping the joints in cotton; later baths (iodin, bog, mud), massage, etc.

10. *Cryptogenetic Septicopyemia.*

Those severe septic diseases in which, unlike puerperal fever and accidental septic wounds, the portal of infection cannot be demonstrated with certainty are included under the designation of cryptogenetic septicopyemia. Frequently, however, it is possible to find neglected injuries, with consecutive phlebitis and lymphangitis, as points of invasion.

The onset occurs with a severe chill, vomiting, and great prostration. The fever is irregular and high, and the pulse frequent. The spleen is enlarged and sweating is free. The sensorium is obscured and a typhoid state may develop.

Repeated chills occur, together with hemorrhages into the skin and the mucous membranes. Abscesses form in glands, lungs, kidneys, liver, spleen, etc. (pyemia), as local manifestations, in addition to symptoms of general intoxication (septicemia). Generally there exists hemorrhagic nephritis. There may be further purulent or serous pleurisy, pericarditis, meningitis, ulcerative endocarditis. The last may be the source of new metastatic embolic infections.

The *diagnosis,* which often at the beginning is difficult, is based upon the repeated chills, the high fever, the hemorrhages, the abscesses, and the endocarditis. As leukocytosis usually attends pyemia, typhoid fever can be excluded by this means. The chills and the failure to demonstrate a primary tuberculous focus tend to exclude miliary tuberculosis.

The *prognosis* is dubious.

The *treatment* is symptomatic. Baths may be employed and stimulants administered. Large doses of alcohol are well borne and prove useful.

11. *Croupous Pneumonia* (Fibrinous, genuine pneumonia).

It is probable that croupous pneumonia can be caused by various cocci, although it has been determined with considerable certainty that the diplococcus described by Fränkel (see p. 122) is the active agent in the majority of cases. The streptococcus pyogenes, and possibly other microörganisms, also may be causative factors.

The disease may involve only one or several lobes (wandering pneumonia). The morbid process consists in the extravasation of a fibrinous exudate into the alveoli, which subsequently undergoes coagulation.

The onset is attended with a chill, high fever, pain in the side, and cough. The fever is continued. The sputum is bloody, viscid, and rust-colored. An eruption of herpes often appears on the lips and about the nose. Marked leukocytosis exists, and the spleen is enlarged.

Over the diseased lung there is at first a slightly tympanitic note on percussion, with crepitant râles during inspiration (stage of congestion). Later there is profound dulness, with bronchial breathing and increased vocal fremitus (stage of hepatization). In the third stage (that of resolution) the dulness clears up, and gives away to a distinct tympanitic note, with numerous moist râles. The course of the fever is indicated in the temperature-chart shown in Fig. 42.

On the fifth day there often occurs a decline in the temperature (see Fig. 43), designated a pseudo-crisis. The true crisis occurs on the seventh or ninth or eleventh day.

Complications.—Dry and exudative pleurisy, empyema, endocarditis (especially the ulcerative variety), heart-failure (especially in alcoholics, the aged, etc.).

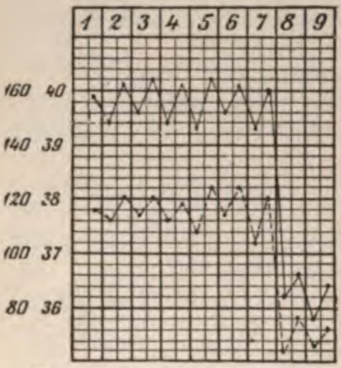


FIG. 42.—Continued fever, with crisis on the seventh day, in a case of croupous pneumonia.

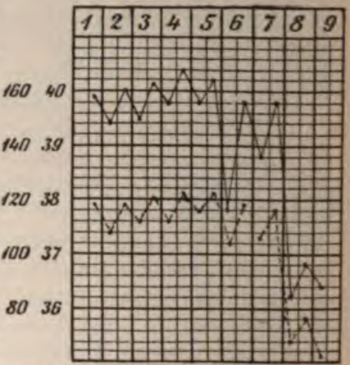


FIG. 43.—Pseudo-crisis on the fifth day in a case of croupous pneumonia.

Abnormal Terminations.—Resolution may be protracted for from two to five months, the fever declining gradually, and the lung may undergo contraction or caseation or become the seat of abscess or gangrene.

Prognosis.—The state of the pulse and of the general strength is an important prognostic indication. In children the prognosis is good.

Diagnosis.—To be noted are the acute onset, the sputum, the leukocytosis (as against typhoid fever), the absence of a sense of resistance and the presence of increased vocal fremitus over the dulness (as against pleurisy). (See differential diagnosis, p. 167.)

Treatment.—Ice-bag, cold drinks, nourishment with milk and eggs (albumin), cold packs, mustard-plaster, injections of morphin, later digitalis and stimulants.

12. *Epidemic Cerebrospinal Meningitis.*

This infectious disease may be sporadic as well as epidemic. It is dependent upon the activity of the diplococcus of Fränkel, which gains entrance through the nose, injuries, the lymph-stream, and the blood-stream, and whose presence and development excite inflammation in the pia-arachnoid of the brain and spinal cord. As a result there develops first a serous, then a purulent, exudate in the interstices of the pia, especially of the convexity of the cerebral hemispheres.

The disease sets in with considerable abruptness, frequently in young persons with general malaise, followed by severe headache, vomiting, and chill. The symptoms become rapidly aggravated. Herpes appears upon the lips, the fever is high and continued, leukocytosis is marked, stiffness and pain upon movement appear in the muscles of the neck (from irritation of the upper cervical nerve-roots), and consciousness is obscured. Delirium, signs of cortical irritation (convulsions), also paralyses (monoplegia, hemiplegia), and hyperesthesia supervene; and finally profound coma, trismus, opisthotonos, retention of urine, constipation, Cheyne-Stokes breathing. In the most severe cases death takes place within a few (usually less than eight) days. In less severe cases the course may be protracted to eight weeks and more. Recovery is, however, possible from the more severe varieties of the disease.

Sequelæ.—Deafness, from a lesion of the auditory nerve; blindness, headache, paralyses, abscess of the brain.

Diagnosis.—To be excluded are typhoid fever (gradual onset, absence of herpes and of leukocytosis); pneumonia (sputum, pulmonary symptoms); pyemia (pus-collection, no rigidity of the neck); tuberculous meningitis (basal symptoms, demonstration of tuberculosis elsewhere); purulent meningitis (pus-accumulation, not epidemic).

Treatment.—Rest, cold pack (baths are painful), ice-bag, withdrawal of blood from the mastoid region, calomel, gr. iv, thrice daily; cantharidal plaster to the nape of the neck, mercurial inunctions, antipyretics, narcotics.

13. *Erysipelas.*

Erysipelas is caused by the streptococcus pyogenes, which undergoes multiplication in the lymph-spaces of the affected part. The portal of entry is often the nose (catarrh, injury), the tonsils (angina), the ear.

The disease sets in with chill and high fever and symptoms of profound general intoxication (headache, pains in the extremities, obscured sensorium, vomiting). The local symptoms include redness, swelling, painfulness of progressive extent (wandering erysipelas) upon the face (edema of the eyelids), the scalp, the neck, etc. The cutaneous lesion is ~~circumscribed~~ by a ~~well-defined raised~~ border, and it becomes the seat of vesicles and gangrene. The temperature may be hyperpyretic. Recovery may be attended with loss of hair.

Prognosis.—Grave in cases of heart-failure (alcoholics, old age).

Treatment.—Poultices (often better than ice), sustained nutrition, cold baths, stimulants, limitation by adhesive strips, ichthyol dressings, etc.

C. Actinomyces-infections.

Actinomycosis of lungs, glands, and bone is described at p. 125, and illustrated in Plate 10, Fig. 6.

The *treatment* is either surgical or consists in the administration of potassium iodid.

D. Spirillum-infections.

14. *Relapsing Fever.*

The cause of relapsing fever is the spirocheta of Obermeier (see p. 125), which can be found in the blood at the height of the paroxysm (see Plate 5, Fig. 4). The disease is exceedingly rare in the United States.

The *period of incubation* is from five to seven days. The disease sets in with chill and high fever. The fever is of continued type. The constitutional symptoms include headache, pains in the back, and prostration. The spleen is enlarged. Jaundice is frequent. The sensorium remains clear. On the fifth day the temperature declines by crisis (see Fig. 44). After a free interval of several days a second usually slighter paroxysm occurs, and recurrence may take place as often as five times.

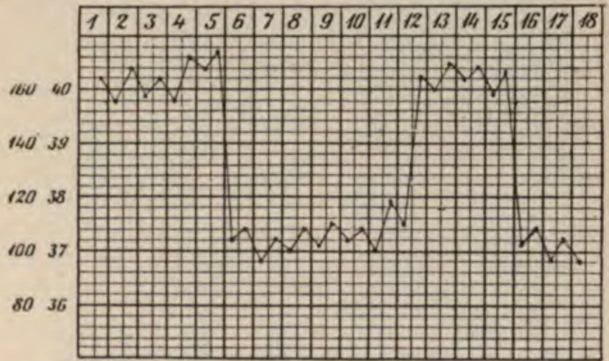


FIG. 44.—Temperature-chart from a case of relapsing fever.

The *diagnosis* is to be made by finding spirilla in the blood.

The *treatment* is symptomatic.

E. Plasmodial Infections.

15. *Malarial Fever* (Quotidian, tertian, quartan intermittent).

This is an endemic infectious disease. Malarial plasmodia are described on p. 130, and are depicted in Plate 5, Figs. 5 and 6.

The *period of incubation* is from seven to twenty days. The *onset* is usually unattended with noteworthy *prodromes*, being marked by a chill and high fever. The temperature may reach 41.5° C. (106.7° F.) in the course of several hours. The chill is replaced by feelings of intense heat. Frequently an eruption of herpes takes place. The skin assumes a yellowish hue. The pulse is frequent and dicrotic, often monocrotic (see p. 70). Headache is present and sometimes delirium. The spleen is greatly enlarged and palpable. In the third stage defervescence occurs by crisis, with profuse perspiration.

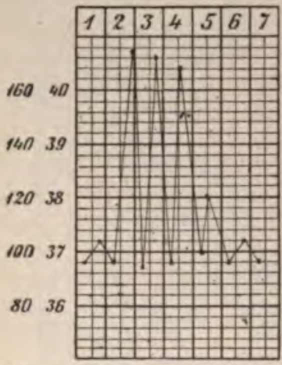


FIG. 45.—Temperature-chart from a case of quotidian malarial fever.

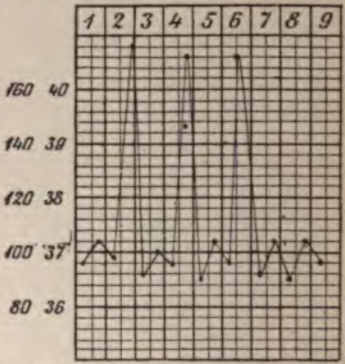


FIG. 46.—Temperature-chart from a case of tertian intermittent fever.

The paroxysms last usually from six or eight to ten hours and are repeated either daily (quotidian, see Fig. 45), or every second day (tertian, see Fig. 46), or every third day (quartan, see Fig. 47). Frequently the paroxysms do not recur always at the same hour of the day, but usually every succeeding paroxysm occurs from two to three hours earlier than the preceding paroxysm (anticipating type), or later (postponing type).

Atypical Varieties.—*Pernicious* malarial fever occurs especially in profoundly malarial regions (Italy, the valley of the Danube). *Masked* malaria (fever atypical or wanting, severe intermitting neuralgia). Chronic malarial *cachexia* (following repeated acute outbreaks, with chronic enlargement of the spleen).

Diagnosis.—The febrile paroxysms alone are typical of the disease (the chills attending pyemia are not dissipated by the administration of quinin). Atypical varieties may be confounded with latent tuberculosis. The detection of plasmodia in the blood is absolutely conclusive.

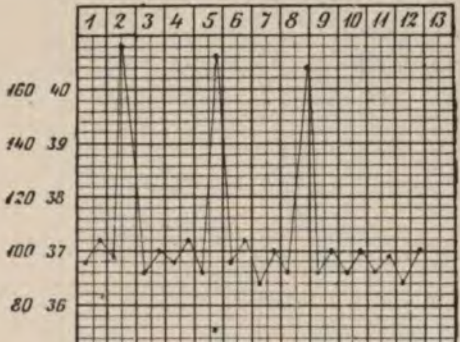


FIG. 47.—Temperature-chart from a case of quartan intermittent fever.

Treatment.—Quinin sulphate, gr. xxv–gr. xxx six hours before the expected attack (causes destruction of the germinating plasmodia). In chronic cases arsenic should be administered and baths directed.

F. Infectious Diseases of Undetermined Origin.

In a number of the diseases about to be described (measles, smallpox, whooping-cough, cholera morbus, dysentery, etc.) bacteria, etc., have been repeatedly found to which causative activity has been attributed.

The *acute exanthemata:* these constitute the most frequent diseases of childhood, recovery from one attack of which confers immunity to subsequent infection with the same disease.

16. *Morbilli* (measles).

The *period of incubation* is ten days, and toward its close *prodromal symptoms* (cough, coryza, vomiting) appear for two or three days.

The disease sets in with a chill, high fever (Fig. 48), and vomiting. Conjunctivitis, photophobia, cough, coryza, are added. The exanthem appears on the third or fourth day, with elevation of temperature. It consists of macules in geographic arrangement, somewhat raised like wheals, and it appears first upon the forehead. The temperature begins to decline by lysis on the sixth day. Desquamation of bran-like scales sets in between the sixth and the tenth day.

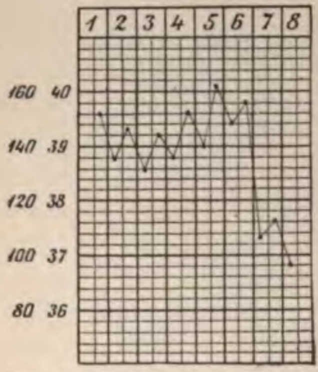


FIG. 48.—Temperature-chart from a case of morbilli.

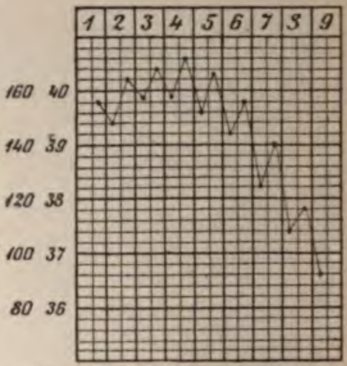


FIG. 49.—Temperature-chart from a case of scarlet fever.

Complications and Sequelæ.—Hemorrhagic exanthem, bronchopneumonia, atelectasis, suppuration of the middle ear, conjunctivitis, croup, tuberculosis.

Treatment.—Baths, packs, milk, eggs, expectorants, and if necessary antipyretics.

17. *Scarlet Fever.*

The *period of incubation* is from two to seven days. The *prodromes* occupy from one to two days and the most constant is vomiting. The attack sets in with a chill and rapidly ascending temperature (Fig. 49). The symptoms point to profound general infection. Angina is present and occasions difficulty in deglutition. The papillæ of the tongue are prominent and give rise to the so-called strawberry tongue. On the second day the eruption appears on the face and neck, the chin escaping, at first as small disseminated red spots, later becoming confluent and presenting a scarlet hue. After the fourth day the temperature declines by lysis. Desquamation occurs in large sheets and continues for from five to fifteen days.

Complications and Sequelæ.—Acute hemorrhagic nephritis (edema), diphtheria (usually fatal), otitis media, endocarditis, encephalitis, articular rheumatism, glandular abscesses, etc.

Treatment.—Baths, douches, packs, gargles, acid drinks, stimulants, symptomatic.

18. *Rubeola* (Rötheln).

This is a mild infectious disease lasting from two to three days. It is attended with a red macular exanthem. The fever is slight. Conjunctivitis may be present, together with slight catarrhal manifestations. Desquamation does not occur.

19. *Variola* (smallpox).

The *period of incubation* is from ten to thirteen days. *Prodromes* may continue for two or three days. The *stage of invasion* is characterized by chill, high fever, symptoms of general infection, pains in the sacral region, cutaneous erythema, petechiæ. In the *eruptive stage*, which occurs between the fourth and the ninth day, the exanthem is present upon the face, upon the body, and also upon the mucous membranes. It appears at first as red spots that soon become elevated into small nodules. These in turn are transformed into vesicles, which become filled with pus and form pustules. In the *suppurative* *stage*, which occurs from the ninth to the twelfth day, the temperature again rises and is of an intermittent, septic, suppurative type. There is also delirium and there may be metastatic suppuration. In the *stage of desiccation* the pustules discharge their contents and dry up, while the fever declines by lysis. Decrustation goes on for from four to six days, leaving cicatrices at first bluish pink, but subsequently becoming snow-white.

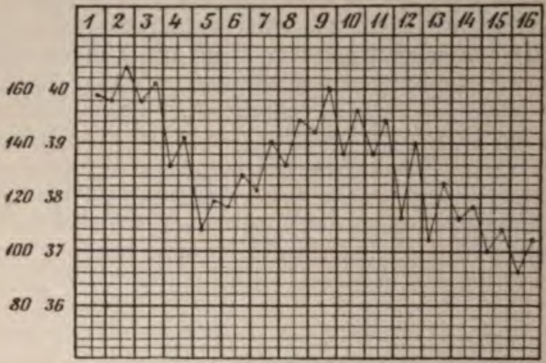


FIG. 50.—Temperature-chart from a case of smallpox.

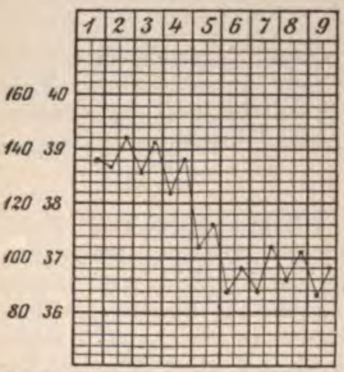


FIG. 51.—Temperature-chart from a case of varioloid.

Modifications. — *Varioloid* is a mild form of variolous infection, unattended with suppuration. It occurs in those who have been vaccinated or have passed through an attack of smallpox. In hemorrhagic variola hemorrhage takes place into the pustules, the skin, and the mucous membranes. This is the severest form, and is known as black smallpox.

Complications and Sequelæ.—Abscess-formation, gangrene of the skin, diseases of eye and ear, pneumonia, nephritis, myelitis.

Treatment.—Baths, symptomatic, stimulants, prophylaxis, vaccination every six to ten years.

20. *Varicella* (chicken-pox).

This is a mild infectious disease occurring almost exclusively in children. It is unattended with noteworthy prodromata. Small vesicles appear in irregular order upon the skin and mucous membranes, replacing previous red spots, and dry up in the course of several days. The fever is irregular and may reach 39° C. (102.2° F.). In some cases nephritis may occur as a sequel.

21. *Epidemic Parotiditis* (mumps).

This is a mild infectious disease occurring usually in children. Amid febrile manifestations the parotid, also the submaxillary gland, and at times the testicles, become painfully swollen. The attack lasts several days. Suppuration is uncommon.

22. *Pertussis* (whooping-cough).

This is a common infectious disease among children, and is feared on account of its long duration and its sequelæ.

The *catarrhal stage* lasts from several days to weeks, and is attended with cough and mild febrile symptoms.

The *spasmodic stage* occupies from four to six weeks. It is characterized by the occurrence of paroxysms of cough of a peculiar whooping character (deep, stridulous inspiration, with a short expiratory cough), frequently attended with vomiting and conjunctival hemorrhage. From ten to thirty or sixty attacks may occur daily, and they are particularly frequent at night.

In the *stage of decline* there is gradual subsidence of the paroxysm in both frequency and severity.

Sequelæ.—Bronchopneumonia, pulmonary tuberculosis, anemia, emphysema.

Treatment.—Change of air, much out-of-door life, moist packs, cold ablutions. Each paroxysm attended with vomiting should be followed immediately by the administration of nourishment (milk, eggs, broth, etc.). Bromoform from three or five to ten drops may be given thrice daily. Sozoiodol may be insufflated into the nares and ethereal oils may be inhaled. Bromin, codein, and belladonna may be administered internally.

23. *Cholera Morbus* (cholera nostras).

This occurs frequently in children, but also in adults, and particularly in the summer months, in consequence of the multiplication of streptococci in the intestines (see Plate 11, Fig. 3; Fig. 1, p. 3). The disease sets in suddenly, with severe vomiting and diarrhea. The stools are watery and the pain is colicky. There is thirst, together with profound collapse, heart-failure, anuria. The attack lasts from six to twelve or twenty hours. Convalescence is protracted.

The *diagnosis* may eventually require bacteriologic examination of the stools for cholera-bacilli (see p. 27).

Treatment.—Opium, calomel, bismuth, astringent wine, mucilaginous soups, poultices, stimulants, and possibly infusions of saline solution.

In children, milk should be withdrawn and mucilaginous decoctions and artificial food given.

24. *Dysentery.*

This disorder is characterized by ulceration of the large intestine, following diphtheric destruction, possibly due to the activity of amebæ. After a period of several days of diarrhea aggravation takes place between the third and the fifth day, with the development of severe diarrhea, painful tormina (griping), and tenesmus (bearing down). There is, besides, a sense of chilliness and prostration. The fever is irregular. Collapse may take place. The attack lasts from three to five days. The stools contain mucus, pus, and blood.

Complications.—Nephritis, pneumonia, perforative peritonitis.

The *prognosis* is grave.

Treatment.—Castor-oil, calomel, opium, suppositories, saline infusions, mucilaginous enemata, concentrated liquid diet, stimulants, hot packs, disinfection of the stools.

25. *Typhus Fever* (spotted fever).

The *period of incubation* is from seven to fourteen days.

After a short *prodromal period* (in contrast with typhoid fever) the temperature rises quickly to a high degree, with a feeling of coldness.

The fever is continued, the temperature ranging in the neighborhood of 40° C. (104° F.). Vomiting is present, with nervous disturbances and delirium. The spleen is enlarged. From the third to the fifth day a roseolous exanthem appears upon the trunk and the extremities and becomes transformed into petechial hemorrhages. The state is a typhoid one and bronchitis is likely to be present. The attack lasts from two to three weeks. Should death not occur from heart-failure the fever declines rapidly. The mortality from the disease is between 10 and 20 per cent.

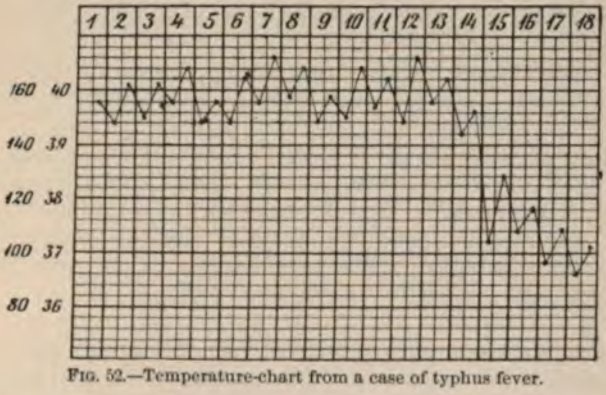


FIG. 52.—Temperature-chart from a case of typhus fever.

Treatment.—Baths, packs, albuminous diet, generous administration of liquids, stimulants.

26. *Hydrophobia* (canine rabies).

Infection occurs through the bites of dogs, wolves, etc. The toxic substance is present in the spinal cord.

The *period of incubation* may extend over many months.

The *prodromal period* covers several days and may be attended with a melancholic state of mind, irritability, anorexia, and insomnia.

In the *hydrophobic stage* spasm of the esophageal muscles occurs when an attempt is made to take food or drink. The glottis is closed spasmodically and breathing is difficult. There are, besides, salivation, delirium, hallucinations. Death often occurs in collapse in the course of several days.

Treatment.—Protective inoculation, cauterization of the wound, narcotics (chloral,·morphin), stimulants.

27. *The Plague.*

The plague formerly occurred repeatedly in Germany as an epidemic disease, being known in the sixteenth century as "black death." It is characterized by a profound septic state. There occurs universal suppurative lymphadenitis (buboes), with hemorrhages. The mortality ranges from sixty to ninety per cent.

28. *Yellow Fever.*

This disease occurs endemically in the tropics, particularly upon the coast in certain regions of America, Africa, and Asia. Transmission takes place through marine intercourse.

The attack sets in suddenly, with a chill, high fever, and vomiting. The extremities are painful. After three or four days jaundice appears. The temperature again rises and hemorrhages may take place into the stomach and bowels, with albuminuria and anuria. The mortality is from 15 to 50 per cent.

Treatment.—Castor-oil, calomel, baths.

29. *Trichinosis.*

This is a parasitic disease due to the invasion of the tissues of the body by embryo trichinæ (see p. 127). The intestinal parasites remain within the bowel for from six to eight weeks. Eight or ten days after infection has taken place the embryos begin their migration.

The first symptoms include gastro-intestinal disorders, with nausea, diarrhea, and febrile manifestations, the temperature rising as high as 40° C. (104° F.). At the beginning of the second week pain is felt in the muscles upon movement and upon pressure. Mastication, deglutition, articulation, and respiration (bronchitis, bronchopneumonia) are painful. There may be also edema of the face and of the extremities and, further, delirium and collapse. Convalescence may be greatly retarded.

Diagnosis.—The painfulness of the muscles is important in distinction from articular rheumatism; the edema in distinction from typhoid fever. Excision of a bit of muscle may be eventually necessary. Examination of the feces often fails to yield conclusive information.

Treatment.—Castor-oil, calomel, glycerin, morphin, sodium salicylate, warm packs.

II. DISEASES OF THE RESPIRATORY ORGANS.

A. Diseases of the Nose.

1. *Acute and Chronic Rhinitis* (coryza).

Acute rhinitis is an infectious catarrhal inflammation of the nasal mucous membrane, attended with mild febrile manifestations, headache, and obstructed nasal breathing. The secretion is at first abundant and mucous, but later purulent. The affection is serious in infants through interference with nursing.

Frequently repeated attacks of nasal catarrh or frequent exposure to moist and cold air, in dusty rooms, etc., leads to the development of *chronic nasal catarrh*. This occurs with especial frequency in individuals with congenital deformities of the nose, such as deflections of the septum, hypertrophy of the turbinate bodies; and in anemic and scrofulous children. Under these conditions the nasal mucous membrane becomes partly hypertrophic and thickened, partly atrophic and attenuated. The secretion drys up into crusts with especial readiness in the atrophic variety. Beneath the crusts ulcerative changes may take place.

Treatment.—Insufflations of menthol; carbolic acid and alcohol, each 10; liquor ammoniæ, 5 (by inhalation); applications of cocain; irrigation with sodium chlorid in cases of acute rhinitis.

Chronic nasal catarrh may be treated with insufflations of europhen, sozoiodol, aristol, boric acid; or irrigation with sodium chlorid, solution of salicylic and boric acids; or with applications of silver nitrate, tannic acid and glycerin, etc.

2. *Ozena* (fetid nasal catarrh).

This disorder occurs as a complication of chronic atrophic rhinitis with crust-formation. The decomposition of the secretion through the activity of putrefactive bacteria brings about the characteristic offensive odor. Frequently the disease is indicative of more deeply seated suppurative processes, such as empyema of the maxillary antrum, of the sphenoid sinuses, caries, etc., which may be detected by the use of the probe in conjunction with aspiration.

Treatment.—Antiseptic irrigation and insufflation, etc. (see special text-books).

3. *Epistaxis* (bleeding from the nose).

This occurs especially in cases of chlorosis and anemia; also frequently in cases of contracted kidney, leukemia, cardiac lesions, and acute infectious diseases. Not rarely there are present upon the nasal septum small telangiectatic areas of the mucous membrane, after cauterization of which the bleeding ceases.

To effect control of the hemorrhage vinegar-water, etc., may be insufflated, or a tampon may have to be employed, if the hemorrhage be severe, passed from behind forward with the aid of a Bellocq cannula. (For further references to diseases of the nasal cavity, see p. 35. For details, reference should be made to special text-books.)

B. Diseases of Larynx, Trachea, and Bronchi.

4. *Acute and Chronic Laryngitis.*

Acute laryngitis is an infectious, mildly febrile, catarrhal disease. Hoarseness is present, in consequence of defective closure of the glottis, and there is irritative cough, with viscid sputum. The vocal bands present a rose-red color and the ventricular bands are swollen and overlie the former—chorditis vocalis superior (see Fig. 53). Often there is slight paresis of the vocal bands, giving rise to the oval glottis (Fig. 54).

Treatment.—Cold applications about the throat; administration of hot milk with Selters or Ems water; codein.

In children the tumefaction of the mucous membrane may give rise to croupy manifestations, dyspnea, pseudocroup.

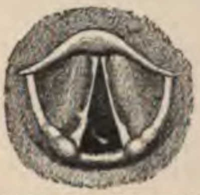


Fig. 53.—Chorditis vocalis superior.

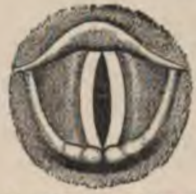


Fig. 54.—Paralysis of the thyro-arytenoids. (Phonation.)

Chronic laryngitis occurs in teachers and singers and in alcoholics. In addition to hoarseness, there is present a troublesome tickling sensation, with a feeling of dryness in the throat. The disease is very obstinate. The vocal bands present a grayish-red color. The follicles are swollen and the mucous membrane is thickened and indurated.

Treatment.—Inhalations (Ems water, Soden water, solutions of tannic acid and alum); applications of silver nitrate solution, from 3 to 10 per cent.; insufflation of powders; bath cures (Ems, Soden, Reichenhall, etc.).

5. *Edema of the Larynx.*

This is characterized by swelling of the aryepiglo
ligaments, with resulting marked stenosis of the lary
giving rise to suffocative attacks. It occurs in conju
tion with inflammatory and catarrhal disorders of
larynx, with the presence of foreign bodies in the lary
with traumatisms, ulcerations and with general edema.

Treatment.—Incisions into the edematous parts ; trac
otomy.

6. *Tuberculosis of the Larynx* (see p. 134).

7. *Syphilis of the Larynx.*

Of noteworthy importance are only the tertiary lesio
In consequence of the breaking down of gummata d
ulcerations form, and stenosis results from cicatrizati
and secondary contraction.

Treatment.—Mercurial inunctions ; for stenosis, intu
tion, tracheotomy.

8. *Paralysis of the Laryngeal Muscles* (see p. 38).

9. *Spasm of the Glottis* (Laryngismus stridulus).

This condition is characterized by repeated attacks
spasm, with severe suffocative paroxysms, and it occurs
scrofulous and rachitic children and in cases of hyste
and of tabes dorsalis.

Treatment.—Removal of the primary disorder, regu
tion of the diet, cold frictions. In the attack cold douc
may be employed, together with hot enemata and artific
breathing.

10. *Tumors of the Larynx.*

These usually arise from the vocal bands and the v
tricular bands and give rise to disturbances of speech (su
as hoarseness, piping voice, diplophonia), and difficulty
breathing (inspiratory dyspnea, suffocative attacks).

It is important to determine whether the neoplasms
benign (fibroma, adenoma, polypi, circumscribed growt
or malignant (carcinoma, rarely sarcoma : diffuse, oft
ulcerative swelling, lymphatic glandular enlargeme
Partial excision and histologic examination may be nec
sary.

The *treatment* is surgical.

11. *Acute and Chronic Bronchitis.*

Acute bronchitis is an infectious febrile disease of the bronchial mucous membrane of greater or less severity, attended with swelling, hyperemia and increased secretion. The symptoms include irritative cough, expectoration, at first mucous, later purulent, pain in the chest, muscular pain (expiratory muscles of respiration, especially the abdominal and the intercostal muscles). The pulmonary percussion-note is unchanged. On auscultation coarse dry or moist râles are heard in greater or less abundance in accordance with the extent of the morbid process, together with prolonged expiration. In cases of capillary bronchitis, with involvement of the smallest bronchi, moist râles with wheezing are heard.

Treatment.—Abundant administration of liquids (Ems water, milk, tea), moist packs, warm baths, codein, expectorants.

Chronic bronchitis results from constant exposure to dusty air, as in factories, etc. It often persists for years and in consequence of the frequently repeated paroxysms of coughing leads to emphysema of the lung and circulatory disturbance.

Catarrh of the apices of the lungs, especially if unilateral, is suggestive of tuberculosis. Hypostatic bronchitis occurs in conjunction with cardiac lesions and diseases of the kidney.

Bronchoblennorrhea is attended with copious diffluent expectoration, the sputum when permitted to stand separating into layers (see p. 51). *Dry catarrh* is attended with annoying cough and tough mucous sputum and respiratory difficulty.

The results of auscultation vary in accordance with the character and amount of the secretion: numerous medium-sized moist râles, especially over the lower lobes, or coarse, snoring, whistling dry râles (sonorous and sibilant).

Treatment.—Change of air; sojourn at sea, in southern

climates, at altitudes, inhalations of oil of pines, turpent
mineral waters, moist packs, frictions, expectorants, e

Croupous bronchitis is an uncommon disorder atten
with the formation of membrane upon the mucosa (fib
ous exudation). It is characterized by paroxysms
dyspnea and cough. The expectorated matters cont
bronchial casts, Curschmann's spirals, Charcot-Ley
crystals (see p. 53). There is usually some fever.

Treatment.—Potassium iodid, expectorants, emet
inhalations of lime-water, alkaline waters, etc.

12. *Fetid Bronchitis.*

This results from the decomposition of stagnant sec
tion in the bronchi through the activity of putrefact
bacteria, and is a common accompaniment of bronchi
tasis. There is usually fever, together with abund
fetid sputum that upon standing separates into layers (
p. 51). In the purulent layer so-called Dittrich's pl
(grayish-white particles) are found, together with f
crystals, bacteria, and masses of detritus. Frequently t
fingers are drumstick-shaped, from bulbous thickening
the terminal phlanges. Metastatic suppurative proces
may develop, particularly in the brain.

Treatment.—Oil of pines, turpentine, myrtol by inha
tion.

13. *Bronchiectasis.*

Saccular or cylindric dilatation of the medium-siz
and especially of the smaller bronchi, principally of t
lower lobes of the lungs, as a result of accumulation
secretion may occur in consequence of atrophy and abn
mal yielding of the bronchial walls. The surroundi
pulmonary tissue becomes destroyed, and there occur fr
quently secondary catarrhal infiltration and interstit
contraction. The sputum is abundant, at times bei
ejected in mouthfuls, as in cases of bronchoblennorrh
and of fetid bronchitis. Frequently tuberculosis is
secondary complication. Upon examination there
found dulness on percussion, especially over the low
lobes of the lungs, together with coarse mucous and al

ringing râles. At a later period the percussion-note may be tympanitic, and the breathing bronchial, from the existence of a cavity. At times there may also be hemoptysis and the fingers may be drumstick-shaped.

Treatment.—See chronic and fetid bronchitis (pp. 165, 166).

14. *Bronchial Asthma.*

This is characterized by paroxysms of acute expiratory dyspnea in consequence of spasmodic narrowing of the smaller bronchi. It occurs frequently in nervous individuals, and is sometimes induced through reflex influences, such as diseases of the nose.

The attacks occur especially at night, and are attended with laborious wheezing inspiration and prolonged expiration.

The boundaries of the lungs are extended during the attack (acute dilatation) and percussion yields a strikingly full, loud note. On auscultation numerous faint whistling and creaking râles are heard, with enfeebled vesicular murmur and prolonged expiration.

The sputum is scanty, viscid and mucous, and contains leukocytes, eosinophile cells, Charcot-Leyden crystals, Curschmann's spirals (see Plate 8, Figs. 5 and 6).

Treatment.—Attention should be directed to possible disease of the nose. Cold frictions and a sojourn at sea are to be recommended. During the attack potassium iodid and morphin may be given, with inhalation of ignited stramonium powder or of vapor of pyridin, etc.

C. Diseases of Lungs and Pleura.

15. *Croupous Pneumonia* (see p. 147).

Differential diagnosis between pneumonia and pleurisy:

PNEUMONIA.	PLEURISY (see p. 172).
Onset.—Always acute, with chill.	Usually subacute, less commonly acute; rarely with chill.
Percussion.—At first relative dulness, with tympanitic accompaniment; later intense dulness, without a *sense of* resistance.	From the outset intense absolute dulness, with a sense of resistance.

PNEUMONIA.	PLEURISY.
Auscultation.—Loud bronchial breathing over the area of most marked dulness.	Enfeeblement or absence of br ing over area of most m dulness.
Vocal Fremitus.—Usually increased; enfeebled only with accumulation of secretion.	Always enfeebled.
Bronchophony.—Loud.	Faint; egophony.
Displacement.—Of neighboring organs, slight.	Marked displacement of heart, spleen.
Sputum.—Blood-streaked, rust-colored, prunejuice-like.	Wanting or catarrhal.

In doubtful cases exploratory puncture will yield
clusive evidence. The exudate present should alway
examined bacteriologically. (Inoculation upon agar-a
etc., see p. 26).

16. *Bronchopneumonia* (lobular pneumonia).

Lobular pneumonia is usually secondary to previo
existing bronchitis, and occurs most frequently in
junction with various infectious diseases, such as meas
whooping-cough, smallpox, typhoid fever and influe
and with cardiac lesions. It is frequent in children
in the aged.

The morbid lesion consists in disseminated, sm
bronchopneumonic, cellular infiltrates (without fibrin
exudation and free from red blood-corpuscles), especi
in the lower lobes (through accumulation of secreti
The fever is irregular and there are sweats and diffic
in breathing. Over the affected areas the percussion-
is impaired in greater or less degree, and numerous m
râles can be heard, together with bronchial breathing.

Treatment.—Baths, cold sponging, douching, m
packs, milk-diet, expectorants.

17. *Atelectasis.*

This condition results from the absorption of air f
portions of lung whose bronchi are occluded by secret
In consequence the affected portion of lung becomes
lapsed and free from air. The condition often accomp
ies the bronchitis complicating measles and whoopi
cough. Atelectasis may occur further when the lung

compressed by pleuritic exudate, or as a result of hydrothorax, pneumothorax, or the presence of tumors, etc.

The breathing is accelerated and retraction of the chest-wall occurs in inspiration. Percussion over the atelectatic areas yields dulness.

Treatment.—See Bronchopneumonia. Respiratory gymnastics, treatment of the primary disorder.

18. *Pulmonary Emphysema.*

A distinction is to be made between simple increase in the size of the lung (acute dilatation) and true emphysema. In the latter condition permanent inspiratory distention leads to atrophy of the alveolar septa (rarefaction of the lung-structure). Emphysema is caused principally by chronic bronchitis, the constant cough and obstruction to expiration leading to loss of elasticity of the lungs. It occurs also in conjunction with whooping-cough, asthma, etc.

In consequence of the increased resistance in the pulmonary capillaries (which are eventually destroyed), there results stasis in the pulmonary circulation, with consecutive dilatation and hypertrophy of the right ventricle.

The principal symptoms are shortness of breath and cyanosis, labored, greatly prolonged expiration, marked prominence of the accessory muscles of respiration (sterno-cleido-mastoid, etc.), cough and embarrassed expectoration. The boundaries of the lungs reach anteriorly to the seventh or the eighth rib (the liver-dulness being in consequence diminished), posteriorly to the twelfth dorsal vertebra. The cardiac dulness is entirely obscured by the pulmonary resonance when percussion is practised with the body bent forward. On auscultation physical signs of chronic bronchitis are elicited, with markedly prolonged expiration. Epigastric pulsation is visible, and the pulmonary second sound is accentuated. The demonstrable respiratory mobility increases in accordance with the severity of the disturbance. The emphysematous thorax is described on p. 13.

Complications.—Derangements of compensation of the right ventricle, pneumonia.

Treatment.—See Chronic Bronchitis. Potassium iod
later digitalis, pulmonary gymnastics, pneumatic chamb
compression of the chest, etc.

19. *Pulmonary Edema.*

Acute paralysis of the heart, and particularly of t
left ventricle, is attended with transudation of hem
rhagic serous fluid into the pulmonary alveoli. This co
dition occurs commonly immediately before death.

Symptoms.—Increasingly labored, noisy (rattlin
breathing, cyanosis, embarrassed expectoration of blood
serous, frothy sputum. On auscultation numerous me
ium-sized and large moist râles are heard over the enti
lung, totally obscuring the respiratory murmur.

Treatment.—Stimulants, injection of camphor and eth
(from three to five large syringefuls, one hourly), mustar
plaster, venesection.

20. *Pulmonary Tuberculosis* (see p. 132).

21. *Pulmonary Gangrene.*

Putrid decomposition of portions of lung (sequestr
tion) may occur as a complication of aspiration-pne
monia, perforation of empyemata; abscesses, fetid bro
chitis, pyemic metastases (septic emboli), perforation of t
esophagus into the bronchi (traction-diverticula), diabet
mellitus, etc.).

Symptoms.—Irregular, high fever, dyspnea, pain in th
side. The sputum is abundant, offensive, and on stan
ing separates into three layers. In it may be found ga
grenous shreds of lung-parenchyma, crystals of fatt
acids, detritus, lung-pigment, fat-drops.

Elastic fibers are wanting, from the presence of a solv
ent ferment (see Plate 9, Fig. 2).

There is dulness on percussion (frequently simultaneou
pleurisy), with bronchial breathing and râles.

Complications.—Purulent, putrid pleurisy, pneum
thorax, pericarditis, metastatic abscess in the brain
etc.

Treatment.—See Fetid Bronchitis. Operative inter
vention may be necessary.

22. *Pulmonary Abscess.*

This may occur as a complication or sequel of pneumonia, infarction, traumatism and embolic processes.

There are present symptoms of compression of the lung (dyspnea, irritative cough, pain in the side) and febrile manifestations (chills). There is dulness on percussion over the affected area, with enfeebled bronchial breathing and râles. Perforation may take place into a bronchus (purulent sputum, elastic fibers—see Plate 9, Fig. 1), into the pleural cavity (empyema), etc. A conclusive diagnosis is to be reached only through exploratory puncture.

23. *Pneumonokonioses* (diseases due to the inhalation of dust).

The affections of this group occur in those who are compelled constantly to inhale mineral or vegetable dust, the fine particles being deposited in the lung-structure. In coal-miners the condition is known as anthracosis; in stone-workers as chalicosis; in iron-workers as siderosis. Those who work in tobacco also may inhale the dust of tobacco. Persons thus affected exhibit symptoms of chronic bronchitis, as well as a diminished resistance to secondary infection, such as pneumonia and more especially tuberculosis.

24. *Pulmonary Embolism* (hemorrhagic infarction).

If an embolus gain entrance into a pulmonary artery it may, if it be sufficiently large, cause immediate death; smaller emboli occlude only the narrower vessels and give rise to hemorrhagic infarction of the area normally supplied with blood by the obstructed vessel (venous stasis, with expression of air). The embolus may be derived from the right heart, in consequence of dilatation; or from the veins of the body, in consequence of phlebitis, etc.

The *symptoms* include sudden dyspnea, irritative cough, and pain in the side. The sputum is bloody and fever is present. On physical examination the pulmonary resonance is impaired over the site of embolism and the breathing is bronchial, while râles may be heard over a s area, though frequently they are wanting.

Treatment.—Morphin, digitalis, ice-bag, rest in a fortable position.

25. *Pleurisy.*

Etiology.—The rheumatic variety is considered a 146; the tuberculous variety at p. 135. The affec occurs in conjunction with pneumonia, pulmonary inf tion, gangrene, abscess-formation, glandular suppurat caries of the ribs or vertebral column, etc.

A distinction is made between *dry pleurisy,* atten with the formation of a fibrinous exudate upon the viously smooth surface of the pleura (which is a particul common association with tuberculosis), and *exudative p risy,* attended with extravasation of a serous, albumin or serofibrinous, serocellular, purulent, hemorrhagic putrid exudate, in accordance with the nature of causative agent.

The *onset* is frequently subacute, less commonly ac with chill and severe pain in the side (which disappe however, as the exudate forms, together with disapp ance of the friction). Cough is severe and dyspne present in consequence of the pain. There is no expe ration.

On *physical examination* dulness on percussion is wa ing in cases of dry pleurisy. On the other hand, ther distinct creaking pleuritic friction, synchronous with breathing and occurring in jerks, also palpable with hand and usually observed over the lower lobe.

When a pleuritic effusion has taken place there is tense dulness, with a sense of resistance and enfeeb breathing, frequently also crepitant râles from compress of the lung; but subsequently there are no adventiti sounds. Vocal fremitus is diminished and egophon present. Above the level of the exudate, especially front, the percussion-note is distinctly tympanitic, in c sequence of relaxation of the lung. In cases of rig sided effusion the liver is displaced downward and heart toward the left, while in case of left-sided effusion Traube's semilunar space is obliterated (the normal tympanitic note being replaced by dulness) and the heart is displaced toward the right and upward.

At the beginning of the attack the patient prefers to lie upon the unaffected side to avoid pain; later, upon the affected side in order to breathe more freely with the healthy side. The affected side may bulge and be behind the other in movement.

The upper boundary of dulness usually is oblique from above and behind, downward and forward, in consequence of the development of the effusion in the dorsal decubitus; and no alteration in the level takes place with change of posture (in contrast with seropneumothorax), in consequence of fibrinous adhesions. During the stage of absorption the level of the exudate regularly remains highest in the axillary region, declining anteriorly and posteriorly (Ellis-Damoiseau curve).

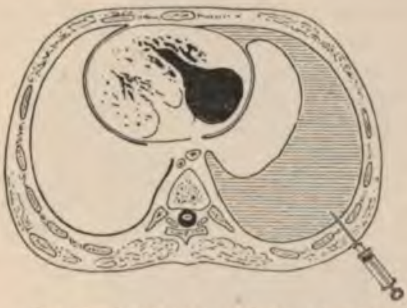


FIG. 55.—Right-sided exudative pleuritis.

The variety of pleurisy is determined by means of exploratory puncture, together with microscopic and bacteriologic investigation (see p. 49 *et seq.*). If the exudate be sterile, the fact is suggestive of tuberculosis. Under other conditions pyogenic cocci, diplococci, etc., are found.

Cure of the pleurisy takes place through absorption of the exudate (increased elimination of urine), and contraction of the affected side (see Fig. 56), with thickening and induration.

Diagnosis.—See pp. 167, 168.

Treatment.—If the level of the exudate reaches anteriorly to that of the second intercostal space, puncture should be made at once. After careful cleansing of the skin with soap, alcohol, and 5 per cent. solution of carbolic acid, a suitable needle previously sterilized by boiling is introduced rapidly and forcefully in the sixth or seventh intercostal space in the scapular line. The exudate is permitted to escape through a rubber tube into a receptacle partially filled with water, the flow being interrupted from time to time. From 1500 to 1800 cu. cm. may be readily evacu-

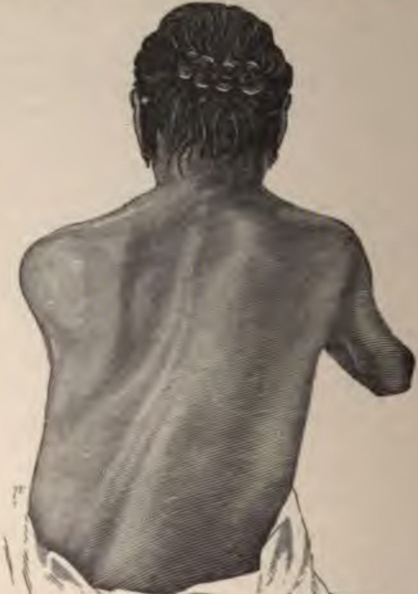


FIG. 56.—Left-sided pleuritic contraction.

ated in this manner, and the wound of puncture is closed with a bit of adhesive plaster. Even in the absence of the foregoing absolute indication early aspiration of the effusion often exerts a favorable influence. It is further to be employed when absorption is tardy. There may be used also mustard-paper, moist packs, ice-bags, diuretics, antirheumatics, morphin.

In cases of empyema operative procedures are indicated.

26. *Pneumothorax.*

Entrance of air into the pleural cavity may occur in consequence of injuries to the chest, of rupture due to a pulmonary cavity, tuberculosis, gangrene, abscess of the lung, etc. In accordance as the opening is constantly patulous or becomes so only with inspiration, or is again closed, the condition is designated as open or valvular or closed pneumothorax. If fluid also, as serum or pus, is present at the same time in the pleural cavity, the condition is designated as seropneumothorax or pyopneumothorax respectively.

In consequence of the entrance of air the corresponding lung is subjected to marked compression, as a result of which there occur suddenly severe dyspnea, sharp pain, often a condition of collapse, fall of temperature (for instance in tuberculosis). The affected side is greatly distended and immovable during respiration. The percussion-note is strikingly deep and full, but as a rule not tympanitic. On percussion with plexor and pleximeter a metallic note is elicited (see p. 30). The vesicular murmur is wanting (in cases of open pneumothorax the breathing in amphoric). If, in addition to air, fluid also is present, the level of the fluid changes temporarily with change in posture. Liver and heart are displaced. Exploratory puncture may be required.

Treatment.—This is to be governed by the nature of the primary disorder. Traumatic pneumothorax may be completely absorbed. Eventually operative evacuation may be required, as in cases of empyema.

27. *Hydrothorax.*

Bilateral accumulation of fluid transudate (containing a deficiency of albumin) in the pleural cavity, especially upon the left side and below, occurs in conjunction with acute nephritis and conditions of stasis attending diseases of the heart.

Hydrothorax gives rise to symptoms of compression of the lungs similar to those due to pleuritic exudation, except that they are bilateral, rather than unilateral: dulness on percussion, with a sense of resistance and enfeeblement of respiration. The fluid removed by aspiration has a specific gravity below 1015, while that of the pleuritic exudate is above 1017.

Symptoms.—Difficulty in breathing, irritative cough, cyanosis.

Treatment.—Diuretics, diaphoretics, hot packs.

Hemothorax results from hemorrhage into the pleural cavity, as from rupture of an aneurism or traumatic rupture of blood-vessels.

28. *New-growths in the Chest.*

Among the new-growths that may occur within the thorax are carcinomata and sarcomata of the lungs and pleuræ as well as of the mediastinal glands (primary and metastatic), gummata, echinococcus of the lung.

The *symptoms,* which at first are equivocal, are essentially as follows: increasing difficulty in respiration, irritative cough, pain. Later, there occur abnormal prominence of the chest and irregularly limited areas of dulness, with absence of respiratory murmur and symptoms of displacement of viscera. There will also be evidences of compression. Distention of the cutaneous veins (as well as those upon the arm of the corresponding side), edema of the chest-wall, of the neck, the face, difficulty in deglutition (compression of the esophagus), neuralgic pain (from pressure upon nerve-plexuses) and paralysis (of the arm), bronchial stenosis, paralysis of the recurrent laryngeal nerve (see p. 39).

If the tumors be malignant, there will be also cachexia and enlargement of lymphatic glands. Frequently hemorrhagic pleurisy is a complication, with the presence possibly of carcinoma-cells in the exudate. In cases of echinococcus rupture into a bronchus may take place (see Plate 21, Figs. 7 and 8).

Treatment.—Arsenic, mercurial inunctions, operation. The prognosis is dubious.

III. DISEASES OF THE CIRCULATORY ORGANS.

A. Diseases of the Heart.

1. *Acute Endocarditis.*

This occurs seldom as a primary disorder, but usually as a secondary infection (see p. 145) in cases of rheumatic polyarthritis, following attacks of scarlet fever, measles, diphtheria, smallpox, pneumonia, gonorrhea, pyemia. A distinction is made between a *benign* (*verrucose*) and a *malignant* (*ulcerative*) variety. Both are attended with fibrinous deposits upon the endocardium, in which the respective causative agents are found (pyogenic cocci, diplococci, gonococci, etc.). In the benign variety the fibrinous deposits take the form of small nodular vegetations; while in the malignant variety there form floating coagula and, in consequence of necrosis, ulceration, rendering possible embolic distribution of the infectious material. Exacerbations are also not rare in benign endocarditis (*recurring* variety).

The symptoms, apart from the primary disorder, are often but slight: high fever and palpitation of the heart. Objectively there are tachycardia, a systolic murmur of varying intensity, a valvular pulmonary second sound, dilatation of the heart. The actual diagnosis is often made only from the sequelæ. In the ulcerative form these are mostly due to the emboli, which, as they are usually septic, give rise to metastatic, pyemic processes, attended with chills. The lesions of the benign variety may either heal perfectly or, through cicatricial contraction, calcification, etc., give rise to shortening of

	MITRAL INSUFFICIENCY.	MITRAL STENOSIS.	AORTIC INS CIENCY.
Hypertrophy	Hypertrophy of the right ventricle; hypertrophy and dilatation of the left ventricle.	Hypertrophy of the right ventricle.	Marked hyper of the left ve
Dilatation	Dilatation and hypertrophy of the left auricle.	Dilatation and hypertrophy of the left auricle.	
Apex-beat	Somewhat increased, displaced toward the left.	Not increased; often displaced toward the left.	Displaced far the left and ward; ma heaving, ext massive ape
Epigastric Pulsation	Usually present.	Marked visible pulsation.	
Murmur	Systolic blowing murmur, in addition to the first sound, at the apex of the heart; second sound faint.	Diastolic, often presystolic, usually faint rumbling or galloping murmur at the apex; first sound very loud.	Loud rushing tolic murn mid-sternum quently also systolic mur the apex (r mitral in ciency), etc.
Sounds	Pulmonary second sound markedly accentuated. 1.	Pulmonary second sound markedly accentuated; often divided. 1.	
Cardiac Dulness	Increase of dulness at first toward the left, later also toward the right (increasing dilatation and hypertrophy of the right ventricle).	Increase of dulness, markedly toward the right, less toward the left (displacement of the left ventricle in consequence of hypertrophy and dilatation of the right ventricle).	Markedly inc toward the the axillary
Pulse	Strong, without peculiarity.	Very small; of slight tension; often irregular and intermittent.	Pulsus celer m running-puls ble capillary (diastolic pa the finger- palpable he pulse.
Miscellaneous	At the apex a systolic thrill (purring tremor) is palpable. This is a very common lesion, and the prognosis is favorable.	Severe cardiac lesion; often associated with other valvular lesions (especially insufficiency); frequently brown induration of the lungs (cardiac-lesion cells, tendency to bronchitis, emboli); marked subjective manifestations; cyanosis.	Abnormal sc over the sma ries (brac radial, arch); double over the f artery, doubl mur (see p. 67 Frequent, fav valvular lesi mains compe for a long Derangement compensation rious import.

1. p. + pressure in lungs.

AORTIC STENOSIS.	TRICUSPID INSUFFICIENCY.	PULMONARY STENOSIS.
Moderate hypertrophy of the left ventricle.	Hypertrophy of the right ventricle.	Marked hypertrophy of the right ventricle.
Somewhat displaced toward the left; usually not greatly increased.	Normal situation.	Displaced somewhat toward the left.
	Present.	Marked.
Sawing, long-continued, diastolic murmur, especially to the right of the upper part of the sternum; the remaining sounds mostly faint.	Systolic murmur at lower extremity of sternum.	Loud systolic murmur to the left of the upper part of the sternum. Systolic thrill palpable.
	Pulmonary second sound not accentuated.	Pulmonary second sound feeble.
Increase of dulness toward the left, later also toward the right.	Increase of dulness toward right, especially also upward.	Marked increase of dulness toward the right; slighter increase toward the left (displacement).
Very slow, tardy; of slight fulness; small.	Usually poorly filled; cardiac-systolic venous pulse in the jugular veins; hepatic venous pulse.	Without peculiarity.
An uncommon cardiac lesion. Frequent attacks of syncope and vertigo (cerebral anemia).	Usually secondary to derangements of compensation of the other and especially mitral valvular lesions; relative insufficiency (see p. 56). Marked symptoms of stasis.	In most cases congenital; often associated with other developmental anomalies of the heart; marked cyanosis; drumstick fingers; death usually at puberty or before.

valves and narrowing of orifices—that is, to valvul lesions of the heart.

Treatment.—Rest, ice-bags, salicylic preparation eventually digitalis.

2. *Valvular Lesions of the Heart.*

Abnormal relations between the valvular apparat and the orifices of the heart are caused by thickenin and shortenings of the valve-leaflets, so that these n longer close adequately; and by cicatricial narrowin and calcification of the orifices. Under the conditio first named there results *insufficiency* of the valve; und the conditions second named, *stenosis* of the orifice.

The pathologic alterations referred to occur in th sequence of acute endocarditis, as well as of arterio sclerosis (syphilis, alcoholism, chronic nephritis); furthe they are in part congenital (especially valvular lesions o the right side of the heart). In accordance with th localization of the lesion at one or more of the fou valves and orifices there result eight varieties of valvula defects (insufficiency of mitral, tricuspid, aortic, and pulmonary valves and stenosis of the corresponding orifices For a consideration of the theory of the most importan valvular lesions reference should be made to p. 55 *et se* The special clinical manifestations attending them are outlined in the preceding table, pp. 178, 179.

Most valvular lesions of the heart occasion symptom during the first period of their existence only in the sequence of unusual strain upon the heart, especially mitra and aortic insufficiency. Advanced cases, as well as th presence together of several lesions, but especially mitra obstruction, even at an early period, give rise to the following symptoms, usually in varying intensity: cardiac dyspnea and cyanosis, continuously or in paroxysms (cardia asthma), sense of oppression, vertigo, headache, circulator disturbances (gastric catarrh, in consequence of venous stasis), palpitation of the heart, cough (hypostatic bronchitis After stasis in the pulmonary circulation has existed for long time brown induration of the lung results, of the ex istence of which the presence in the sputum of so-called cardiac-lesion cells (see Plate 8, Fig. 4) affords an indication. Temporary derangements of compensation (see p. 60) occasion increase of dyspnea and cyanosis, further edema, ascites, hydrothorax, cyanotic kidney (albuminuria), cyanotic liver, cyanotic spleen, emboli (brain, kidney).

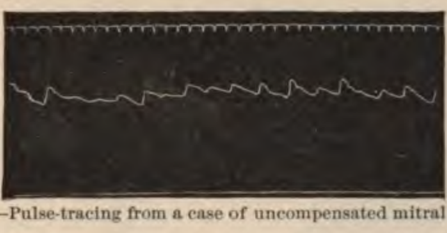


FIG. 57.—Pulse-tracing from a case of uncompensated mitral stenosis.

Diagnosis.—Of importance especially are the situation, the character, and the time of occurrence of the murmur, and, above all, the state of the pulse and the condition of the heart (see table, pp. 178, 179).

Treatment.—For derangement of compensation: powdered digitalis leaves, from 0.1 to 0.15 (gr. jss–gr. ijss) every one and a half or two hours until physiologic effects are produced, as indicated by the pulse; infusion of digitalis (from 1 to 1.5 to 120. [from gr. xv–gr. xx to four ounces] every two hours; 3. to 150. [gr. xlv to five ounces], one-third by enema, twice or thrice daily). Figs. 57 and 58 illustrate the pulse of mitral stenosis before and after the action of digitalis.

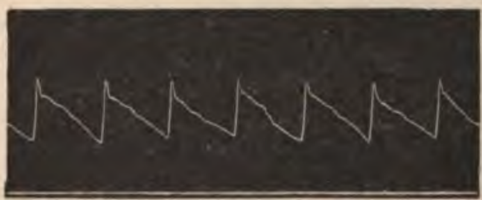


FIG. 58.—Pulse-tracing from the same case as Fig. 57 after administration of digitalis.

If digitalis is not well borne in any form, but not be fore, resort may be had to caffein, 0.2 (gr. iij) four time daily; spartein, 0.0015 (gr. $\frac{1}{40}$) thrice daily; infusio of adonis vernalis, 5. to 150. (gr. lxxv to five ounces) infusion of convallaria majalis, 5. to 150. (gr. lxxv t five ounces); tincture of strophanthus, 10 drops thric daily; diuretics; stimulants; morphin.

For cardiac dropsy calomel, 0.2 (gr. iij from 3 to 5 time daily, following or simultaneously wit digitalis; powdere digitalis leaves 0.1 (gr. ij); calomel, 0. (gr. iij), 5 times daily gargle of potassium chlorate (stomatitis) other diuretics (potassium acetate; theobromin sodio-salicylat [5.–8.—gr. lxxv–ʒi —daily]; squill; potassium and sodium borotartrate); elevated posture; application of bandages; massage of the swollen extremities; aspiration of the ascites, hydrothorax or hydropericardium scarification of the skin, employment of capillary trocar for chronic edema of the skin.

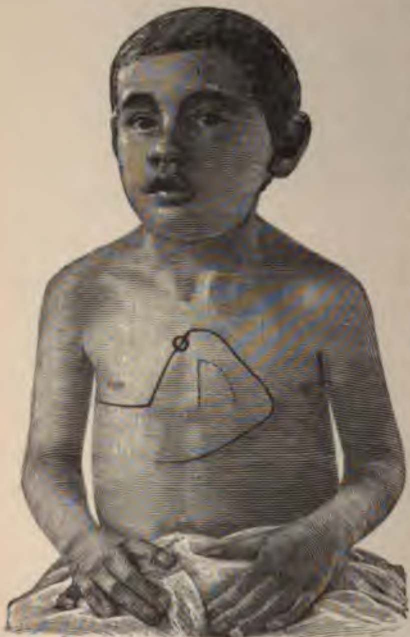


FIG. 59.—Drum-stick fingers in a case of pulmonary stenosis.

In the stage of compensation: avoidance of all excessive physical exercise (bicycling), limitation of the amount of fluid ingested (alcohol); roborant nutrition; cold spongings; regular exercise afoot; baths (Nauheim, Kissingen, etc.).

Congenital Lesions of the Heart.

The valvular lesions (tricuspid stenosis and pulmonary insufficiency) not mentioned in the foregoing table on account of their rarity occur, as well as pulmonary stenosis, usually in consequence of congenital narrowing or defect, generally in combination with other developmental anomalies (patulousness of the foramen ovale, of the duct of Botal, deficiency in the ventricular septum, in the origin of the large vessels).

The accompanying photographs illustrate the conditions presented by two boys suffering from lesions of the kind just named, under observation at the City Hospital of Bamberg. Fig. 59 represents a case of typical pulmonary stenosis (cyanosis; hypertrophy of the right ventricle; displacement of the left ventricle; increased area of cardiac dulness [the normal area of dulness being shown by interrupted lines]; systolic murmur at the base of the heart, heard loudest in the situation of the small circle; drum-stick fingers). Fig. 60 exhibits, in addition to the symptoms of pulmonary stenosis, a band of dulness (patulous duct of Botal [?]) at the upper portion of the sternum above the cardiac dulness. The patient presented, beside, a spastic brachial monoplegia, probably in *consequence* of a previous embolism, perhaps conveyed

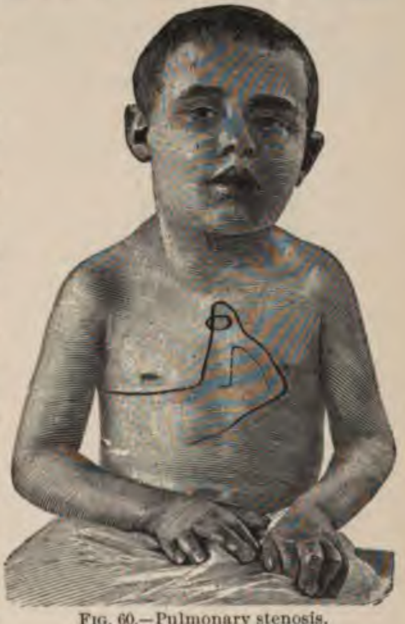


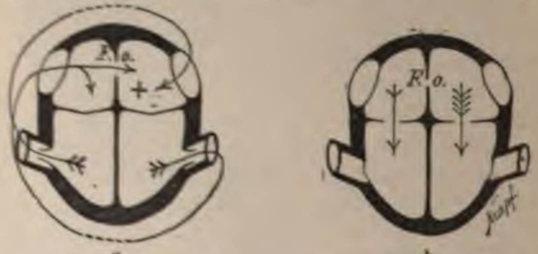
FIG. 60.—Pulmonary stenosis.

Also Club fingers in Tb.

from the right side of the heart through the patul foramen ovale.

When the foramen ovale fails to close (see Fig. 61 and *b*) the venous blood passes in part from the ri auricle, without having passed through the lungs, i the left auricle and ventricle, with resulting admixt of venous and arterial blood, and marked cyanosis.

The *prognosis* is always unfavorable by reason of insufficiency of the arterialization of the blood constan present, and the usually enfeebled development of child (tendency to tuberculosis, to severe derangeme



a. *b.*

FIG. 61.—Patulous foramen ovale. Diagrammatic representation of the cir lation during the systole (*a*) and the diastole (*b*).

of compensation). Children thus affected rarely pass t age of ten or fifteen years.

3. *Idiopathic Hypertrophy of the Heart.*

The designation *idiopathic hypertrophy* is applied cases in which the enlargement of the heart is unattend with the existence of a demonstrable valvular lesion, indicated by purity of the sounds.

Hypertrophy of the left ventricle develops in the sequen of abnormal resistance in the general circulation, of art riosclerosis (rigidity of the arteries) and of contract kidney (increased blood-pressure in consequence of co striction of the small vessels as a result of toxic infl ences); further, in the sequence of excessive ingestion fluids and of food (use of beer), as well as of continu physical overactivity (athletes, gymnasts, oarsmen, etc.).

Hypertrophy of the right ventricle develops in conjunction with increased resistance in the pulmonary circulation, with emphysema, with contracting pleurisy and pneumonia, with atelectasis, with kyphoscoliosis.

The *symptoms* are the same as those that attend valvular lesions. When insufficiency of the muscular structure of the affected ventricle appears, symptoms of derangement of compensation set in (see p. 60); marked dilatation is attended with relative insufficiency (systolic murmur).

Treatment.—It is necessary first to determine the etiologic factor and then to effect its removal. In other respects the treatment is like that of valvular lesions.

4. *Diseases of the Myocardium.*

Fatty degeneration of the myocardium is the usual outcome of diseases of the heart (valvular lesions, idiopathic diseases), associated with hypertrophy and dilatation. The development of this condition is recognized by failure of digitalis to bring about improvement, the diseased myocardium being no longer able to overcome the dilatation. The occurrence of fatty degeneration is induced with especial frequency through the activity of certain toxic substances (alcohol, typhoid fever, scarlet fever, diphtheria); further in consequence of anemia, inanition, etc.

Fibroid degeneration (myocarditis) consists in partial transformation of the muscular structure of the heart into tendinous connective tissue. It occurs in conjunction with disease of the coronary arteries (arteriosclerosis, syphilis). The most important symptoms are attacks of oppression, angina pectoris, embolic processes from mural thrombi, hypertrophy and dilatation of the heart, and especially striking arrhythmia of the pulse, with intermission of the heart's action. Derangements of compensation are always grave. The disorder occurs frequently in association with idiopathic hypertrophy. Rupture of the heart and aneurism-formation may take place.

Treatment (see Cardiac Valvular Lesions).—Potassium iodid, mercurial inunctions, and morphin may be required.

5. *Neurotic Disorders of the Heart.*

Nervous palpitation of the heart (a sense of vig cardiac activity), with or without tachycardia (accele action of the heart), occurs in part permanently in of exophthalmic goiter, in part transiently in cas neurasthenia, of anemia after active physical exe following excitement, in cases of hypochondriasis, relative infrequency (usually only in the stage of dera compensation) in connection with valvular lesions o heart and diseases of the myocardium.

Treatment.—Hardening, cold frictions, systematic ily and mental activity, iron, bromid, etc., cold compr baths (carbonated).

Angina pectoris (*stenocardia*) is characterized by at of intense pain and oppression in the precordium, ating to the arm, the shoulder, and the back, assoc with a condition approaching collapse. It occur nervous individuals, in heavy smokers, in conjun with sclerosis of the coronary arteries and with me tinal tumors.

Treatment.—Cutaneous irritants (mustard), hot or cold compresses, injections of morphin, chloralamid (2 to 3 grams—gr. xxx–gr. xlv); nitroglycerin (0.0 gr. $\frac{1}{20}$—daily); sodium nitrite (2 per cent.), etc.

Fatty Heart.—This designation is applied to a s of manifestations in obese individuals which are la of nervous origin and in part dependent also upon m conditions of the myocardium.

Symptoms.—Sense of oppression, dyspnea, palpit of the heart, painful sensations, etc.

Treatment.—Regulation of the amount of fluid an food ingested. Increase in the muscular activity thr systematic mountain-climbing under medical dire and other forms of gymnastic exercises.

B. Diseases of the Pericardium.

6. *Pericarditis.*

This is usually secondary to rheumatic polyarth scarlet fever, diphtheria; it attends also contracted kidney, alcoholism, pleurisy, phlebitis, tuberculosis, etc.

A distinction is to be made between *fibrinous* (villous deposit), *exudative*, *hemorrhagic* (tuberculosis, neoplasms), and *purulent* (pyemia) varieties.

Symptoms.—Severe, stabbing pain in the precordium, pain in swallowing, sense of oppression, anxiety, dyspnea, irregular fever, enfeebled apex-beat.

Dry Pericarditis.—Loud, rasping pericarditic friction, not synchronous with the action of the heart, especially at the upper portion of the sternum; disappearing with increase in the exudation and returning with its absorption.

Exudative Pericarditis (see Fig. 62).—The area of cardiac dulness is considerably increased in all directions and is of triangular outline (see Plate 49). The apex-beat, if at all palpable, can be felt within the area of dulness and not at its boundary, as it is in connection with hypertrophy and dilatation. The heart-sounds are very faint, but pure. The pulse is accelerated, sometimes irregular.

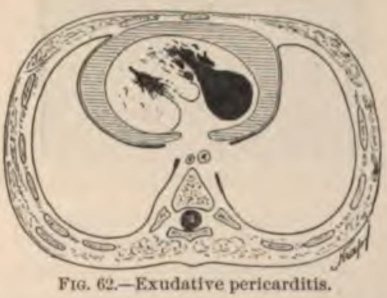


FIG. 62.—Exudative pericarditis.

With the absorption of the exudate there result extensive adhesions of the pericardium, often entirely obliterating its cavity. These occasion disturbances in the activity of the heart (secondary degeneration and atrophy of the myocardium). The condition may be manifested objectively by systolic retraction in the situation of the

apex-beat, with irregular (Fig. 63) and paradoxic
(see p. 68)—diastolic collapse of the veins.

The *prognosis* is dependent upon the etiologic fa
as determined by exploratory puncture in the fifth
costal space to the left of the sternum.

Treatment.—Digitalis, strophanthus, morphin, ice
mustard-plaster, quiet, comfortable posture, diuretics
the exudation be large, aspiration may be practise
purulent, surgical intervention will be necessary.

7. *Hydropericardium* (Dropsy of the Pericardium

This consists in an accumulation of transudate in co
tion with general venous stasis and often due to a ca
lesion in the stage of compensatory derangement ; it
attend acute nephritis and the severe anemias.

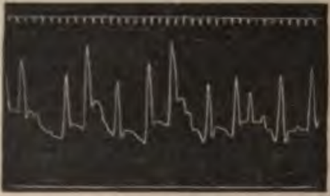


FIG. 63.—Irregular pulse attending pericardial adhesions.

Symptoms.—Dyspnea, sense of anxiety, oppres
irritative cough. Objective manifestations : enlarge
of the area of cardiac dulness, as in exudative peri
itis, enfeebled apex-beat, faint and dull but pure so
no friction-murmur.

Treatment.—In addition to the treatment of the p
ary disorder diuretics, digitalis, calomel, etc., may be
scribed, and finally aspiration may be practised.

Hemopericardium.—A collection of blood in the
cardium takes place in the sequence of injuries, ru
of an aneurism of the aorta and of the coronary art
and of the heart (myocarditis, etc.).

The *symptoms* are like those of hydropericardium,
the addition of suddenly developed anemia, coma, a
fatal termination.

Pneumopericardium.—An accumulation of air in the pericardium takes place in the sequence of perforation of pulmonary or of abdominal abscesses. It gives rise to abolition of the cardiac dulness, with metallic murmurs synchronous with the heart's action, and a metallic note upon percussion with plexor and pleximeter (as in pneumothorax).

The *prognosis* is dubious.

C. Diseases of the Vessels.

8. *Arteriosclerosis.*

Loss of elasticity of the arteries through thickening of the intima, deposition of lime-salts in the intima and media, with secondary fatty degeneration and atheromatous (softening) ulcers in the intima. The condition is manifested by tortuosity and rigidity of the vessels (temporal, radial, brachial). It occurs in advanced life (especially in the aorta, the temporal, the brachial, and the coronary arteries) and at an earlier period as a result of alcoholism, syphilis, lead-poisoning, gout, chronic nephritis, etc.

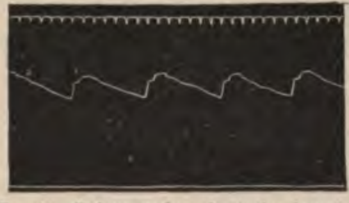


FIG. 64.—Pulsus tardus of arteriosclerosis.

In consequence of the increased resistance in the general circulation, from rigidity of the arteries, the left ventricle undergoes hypertrophy and dilatation. Often systolic murmurs are audible. The pulse is retarded (Fig. 67) and the vessel feels hard.

Sequelæ.—Emboli (fibrinous coagula) and hemorrhages (miliary aneurisms) give rise to apoplexies and disturb-

ances in the cerebral circulation, to vertigo, he
tinnitus aurium, etc. Attacks of angina pectori
occur, from involvement of the coronary arterie
contracted kidney may result. The recognition o
fication of the radial artery does not justify a con
as to the existence of general arteriosclerosis, as i
ment of the aorta may be unattended with similar i
ment of the radial.

The *prognosis* is dubious.

Treatment.—Mild courses of baths at Carlsba
cure at Kissingen. Baths (carbonated, etc.) with c
possibly potassium iodid; otherwise symptomatic.

9. *Aneurism of the Aorta.*

This condition may attend arteriosclerosis and sy
It is mostly situated upon the ascending portion
arch of the aorta. It is manifested by the presen
pulsating swelling in the second and third inte
spaces to the left of the sternum, with dulness on
sion over or adjacent to the upper portion of the st
in cases of large aneurism merging with the cardi
ness. On auscultation a loud vibratory systolic (als
tolic) murmur can be heard. On palpation a thr
be felt. The left ventricle undergoes hypertroph
dilatation. The radial pulses are unequal.

The *prognosis* is dubious.

Treatment.—Potassium iodid, ergotin, compr
perhaps surgical intervention.

IV. DISEASES OF THE DIGESTIVE ORGANS.

A. Diseases of the Mouth and the Pharynx.

1. *Stomatitis.*

Inflammation of the gums is in most cases of inf
or toxic origin.

Catarrhal stomatitis is attended with swelling, re
painfulness of the gums and of the mucous lining
cheeks, and is manifested by marked salivation
breath, decomposition-processes, epithelial desquan

bacterial multiplication. It occurs in conjunction with caries of the teeth, injuries due to chemic substances (acids, tobacco), and with other infectious diseases (measles, syphilis).

Necrotic stomatitis presents an aggravation of the foregoing symptoms, to the point of necrotic destruction of the mucous membrane, with loosening of the teeth, hemorrhages, purulent pasty deposit upon the gums, exquisite pain, marked salivation, swelling of the lymphatic glands. This condition may be transformed into:

Ulcerative stomatitis; after exfoliation of the necrotic tissues superficial ulcers appear, especially at the corners of the mouth, at the junction of the cheeks and the jaws, and at the points where the teeth lie in contact with the buccal mucous membrane. This variety of stomatitis occurs especially in conjunction with mercurial poisoning (calomel, blue ointment, and in artisans).

Aphthous Stomatitis (aphthæ).—This is a disease of the mucous membrane of the mouth, tongue, and palate, dependent in children (through the milk of cows affected with foot-and-mouth disease) upon an as yet undiscovered infectious agent. It is characterized by the presence of small, round, white spots, somewhat depressed and surrounded by an area of redness (fibrinous exudate in the necrotic mucosa).

Bednar's Aphthæ.—These are usually insignificant, atrophic white spots upon the hard palate of nursing infants.

Treatment.—Cleanliness; care of the teeth; withdrawal of mercurial treatment; astringent gargles (tannic acid, alum, borax in solution of from 1 to 5 per cent.); antiseptics (carbolic acid, 1 or 2 per cent., etc.); potassium permanganate, 0.1 per cent.; potassium chlorate, 2 per cent.; solution of hydrogen dioxid, 2 per cent.; applications of tincture of myrrh, tincture of rhatany; in severe cases applications of silver nitrate.

Thrush-deposits (see p. 121) should be treated with applications of solution of borax (5 per cent.), or with solutions of resorcin (1 or 2 per cent.).

2. *Glossitis.*

Parenchymatous glossitis may be attende
formation, fever, tumefaction and intense
the tongue, and also interference with res
to be treated with applications of ice and

Dissecting glossitis is attended with th
numerous fissures and excoriations of t
requires applications of silver nitrate.

Leukoplakia buccalis (geographic tongu
sists in the formation of cloudy, white thi
epithelium upon the tongue, and also u
mucous membrane. The condition is with
and is often mistaken as syphilitic.

Ludwig's Angina.—This is a parenchy
mation of the floor of the mouth, attende
tion of the submaxillary, salivary, and ly
symptoms of profound constitutional di
danger of edema of the larynx.

Treatment.—Early incision.

3. *Angina* (Tonsillitis).

This is an infectious inflammation of
pendent upon the activity of pyogenic c

Catarrhal angina attends scarlet fever,
atitis, etc., and is characterized by rednes
painfulness of the tonsils and of the soft
is no deposit, although fever, headache, e

Lacunar angina is the most common
characterized by the presence of purulen
lacunæ of the tonsils, which do not coales
phatic glands are enlarged.

Necrotic angina is characterized by t
pasty, grayish-white deposits, not involv
The appearances are suggestive of diph
diagnosis should be based upon microscopi
logic examination (see p. 27).

Diphtheric angina is attended with th
white deposits advancing from both sid
palate and uvula until they meet (see p. 1

Parenchymatous angina is characterized by abscess-formation, marked swelling and tension of the tonsil and the palatine arch, usually upon one side only. The condition is attended with great pain, and swallowing is impossible.

Treatment.—Gargles of potassium chlorate (2 per cent.), alum, potassium permanganate, solutions of carbolic acid; or inhalations; ice; cold applications. When suppuration is threatened poultices should be employed, and when an abscess has formed early incision should be practised.

4. *Chronic Hypertrophy of the Tonsils.*

This condition as seen in children is usually congenital. The patients manifest a predisposition to anginas. The condition is attended with interference with breathing and with speech (which is rendered nasal), and with snoring, restless sleep. It is a common accompaniment of scrofulosis.

Treatment.—Nutrition; salt baths; frictions; excision of the hypertrophied parts with the tonsilitome or with the knife.

5. *Dry Chronic Pharyngitis.*

Chronic Pharyngeal Catarrh occurs frequently in association with chronic retronasal catarrh in smokers, alcoholics, anemic individuals, etc. The mucous membrane undergoes atrophy and presents a pale, smooth, glazed appearance, and is frequently covered with tough secretion or crusts. The patient suffers from an annoying sense of dryness, of burning and of scratching in the throat, with constant hawking, and sometimes morning vomiting from heightened reflex irritability.

Treatment.—Nasal douches (sodium chlorid, sodium bicarbonate, alum); insufflation of sozoiodol, tannic acid, silver nitrate (from 0.3 to 0.5 [gr. v–gr. viij] to 10. [ʒijss] of starch); applications of glycerin and iodin, etc.; inhalations; baths (Reichenhall, Ems, Kreuznach, etc.).

Adenoid Vegetations of the Nasopharynx (see p. 36).

Treatment.—Extirpation; galvanocautery.

Retropharyngeal abscess (see p. 81).

Treatment.—Immediate incision.

Paralysis of the Palate; Paralysis of the Phar Both of these conditions sometimes follow diphthe consequence of neuritis of toxic origin. The for attended with the regurgitation through the no fluids swallowed, with a nasal tone of voice and dif or impossibility of gargling; paralysis of the pha with difficulty or impossibility of swallowing.

B. Diseases of the Esophagus.

Inflammatory processes (from catarrh, chemic irri and ulceration (corrosive, tuberculous, syphilitic, decu are not common, and are of significance only with to their sequelæ (stenosis). Diverticula are describ p. 82.

6. *Stenosis of the Esophagus.*

This condition may result in consequence of con sion from without (mediastinal tumors, aneurism aorta, inflammatory processes, caseous bronchial gl of obstruction (by impacted foreign bodies or accu tions of aphthæ), of cicatricial narrowing (syphilitic corrosions, etc.), as well as of carcinoma of the esop (this is the commonest cause).

With the gradually increasing stenosis there difficulty in swallowing, solid articles of food givin to a sense of obstruction and lodgment, with regu tion, eructation, vomiting. The emaciation attai extraordinary degree in cases of marked stenosis finally complete inanition.

The most important information is gained throug use of the sound (see pp. 29, 81), which may also information as to the nature of the obstruction. I be seated at a distance of 23 cm. (9 inches) fro margin of the teeth, it is likely to be dependent glandular tumors, aneurism, etc.; if, however, it be at a distance of 40 cm. (15¾ inches) or slightly less to be attributed to carcinoma of the cardiac orifice stomach.

Cicatricial stenosis is to be recognized from the history. It is often seated high up, at the entrance to the esophagus. A suspicion of carcinoma will be strengthened by the presence of marked cachexia, metastatic deposits, and advanced years.

Treatment.—Surgical intervention will be required eventually (dilatation with bougies after esophagotomy, introduction of nourishment through a gastric fistule). The food should be liquid and mushy, and the tube may be necessary for its introduction. Nutrient enemata (milk, eggs, beef-tea, etc., see Section V, morphin) may be required.

C. Diseases of the Stomach.

7. *Acute Gastritis.*

Acute gastric catarrh is in part infectious and toxic (infectious diseases, cholera morbus), in part a direct result of errors in diet.

Symptoms.—A sense of oppression in the epigastrium, anorexia, nausea, vomiting, eructation, diminished production of hydrochloric acid.

Treatment.—Diet, hydrochloric acid, mild purgatives.

8. *Chronic Gastritis.*

This condition occurs in conjunction with alcoholism, anemia, chronic nephritis, heart-disease (hypostatic catarrh), carious teeth, etc. There is frequently deficient secretion of hydrochloric acid, though not invariably. Other symptoms include tenderness on pressure in the epigastrium, persistent impairment of appetite, moderate emaciation, nausea, eructation (especially upon an empty stomach), vomiting of mucus, heartburn (due to lactic acid from fermentation), hypochondriasis.

Diagnosis.—Carcinoma is to be excluded (cachexia, presence of a palpable tumor, history). Inquiry into the etiology (nephritis, heart-disease, alcoholism, etc.). Examination of the gastric juice (see p. 85 *et seq.*).

Treatment should be directed to the primary disorder. The diet should be regulated, fatty, indigestible foods

being avoided. From time to time a milk-diet sh
prescribed, and cold frictions and massage of the
trium be practised. Attention should be directed t
lation of the intestinal movements, by means of e
of oil, glycerin, or water. Courses at Marienbad,
bad, Kissingen, may be advised.

Dilute hydrochloric acid should be given afte
meal if anacidity is present. Stomachics may be re
and possibly lavage of the stomach with soluti
boric acid, etc.

Phlegmonous gastritis is attended with suppurat
and beneath the mucous membrane, and occurs i
junction with septic infection.

The *prognosis* is dubious.

9. *Gastric Ulcer.*

Chronic ulceration of the stomach is seated mo
the neighborhood of the pylorus and upon the
curvature. It invades the wall of the stomach to
ing depth. The condition may be caused by circu
disorders of various kinds, in consequence of whi
tain areas become anemic and undergo autodigestio
is common in anemic individuals, and especially in w

Symptoms.—Circumscribed pain, occurring s
neously, after the ingestion of food and upon pre
hyperacidity, with the presence of hydrochloric a
the fasting stomach; heartburn; frequent vom
constipation; from time to time hematemesis, wi
ejection of dark blood (blood may also appear i
stools). Recovery may be followed by cicatricial d
ity (hour-glass contraction, pyloric stenosis). Oft
ulcers are multiple. Not rarely carcinoma develop
ondarily upon the cicatrix left by previous ulce
If the ulcer be deep, there is danger of perforativ
tonitis.

Diagnosis.—Circumscribed pain and hematemes
the most important symptoms. Nervous dyspepsia
be excluded. In many cases characteristic sym
are entirely wanting (latent ulcer).

Treatment.—Rest in bed; hot applications at intervals; liquid diet (milk, eggs, broths), sodium bicarbonate (℥j) with bismuth (gr. xxiv) in small quantities; Carlsbad water or salts; large doses of bismuth in mucilaginous vehicles. (See Dietary, Section V.)

Duodenal Ulcer.—This sometimes follows burns of the skin. It is frequently unattended with symptoms, although hemorrhage may take place, the blood appearing in the stools or being ejected by vomiting. Symptoms of peritonitis may appear, with pain, especially in the right hypochondrium.

10. *Carcinoma of the Stomach.*

This occurs mostly in persons between the age of 40 and 60 years. The new-growth is usually a cylindric epithelial carcinoma (diffuse scirrhus, or individual carcinomatous nodules). The neoplasm is usually seated at the pylorus and upon the lesser curvature.

The *symptoms* are of insidious onset. The appetite is impaired and strength fails. There is complaint of gastric derangement, with pain, eructation, heart-burn (due to lactic acid from fermentation). Later there occur vomiting of food, and also stagnation of food. Eventually hematemesis takes place, the blood appearing in masses, resembling coffee-grounds (for demonstration of hemin crystals see Plate 6, Fig. 3). The further progress of the case is marked by increasing cachexia, anacidity, with the presence of considerable lactic acid in the gastric juice. Frequently a tumor is palpable in the region of the stomach, being slightly movable with respiration if not adherent to the liver. Metastases are common, especially in the liver. The duration of the disease may be from six months to a year and a half.

Treatment.—Symptomatic; easily digested food (meat-juice, artificial food, milk, eggs); hydrochloric acid; extract of condurango; possibly gastroenterostomy. If the diagnosis be made early (circumscribed tumors, absence of hydrochloric acid), radical extirpation of the new-growth may be undertaken.

11. *Dilatation of the Stomach.*

This occurs most commonly in conjunction with p stenosis, due to cicatricial stenosis following ulcer the presence of tumors (carcinoma), rarely compr from without (tumors in the neighborhood of the wandering kidney, etc.). The stenosis is at first co sated for through hypertrophy of the muscular wall stomach, but later progressive dilatation occurs. I sequence of the stagnation of food fermentative pro result. Atonic dilatation of the stomach occurs di from over-distention in those who eat and drink sively, and in cases of chronic catarrh. The condit usually associated with hyperacidity.

Symptoms.—Pains in the stomach from two t hours after the ingestion of food; vomiting of amounts at the same time or later; heart-burn; acid tations. The appetite is usually good. Neverth there is progressive emaciation. The pain is miti by the vomiting. The dilated stomach, as well peristaltic activity, is sometimes discernible from t terior. Splashing sounds may be elicited on agitati the body. The enlargement of the viscus may b monstrated by distending it with carbon dioxid, o water (see p. 85 and Plate 51, *b*). The stomach be considered dilated if under these circumstanc lower boundary extends to the level of the umbili below.

Treatment.—See treatment of gastric ulcer. Syste lavage (evening or morning); chopped meat; milk; eggs; massage; electricity. If emaciation be not t advanced (body-weight!): pyloroplasty or gastro ostomy. Eventually nutritive enemata. For the mentative disorders: magnesia and powdered charc small quantities. Irrigation with solution of boric

In reaching a *prognosis* as to the results of surgic tervention it is important to determine whether the osis of the pylorus is benign (cicatricial) or mali (carcinomatous).

Pyloric Stenosis.

	BENIGN.	MALIGNANT.
Duration	Long (from 5 to 15 years).	Short (from 6 months to 1½ years).
Course	With intervals of improvement.	Progressive aggravation.
Tumor	Usually wanting; rarely benign hypertrophy.	Usually present.
Appetite	Almost always good.	Poor.
Hydrochloric Acid	Usually present.	Almost always wanting.
Lactic Acid	Usually wanting.	Always present.
Vomiting	Brings relief.	Causes exhaustion.

12. *Nervous Dyspepsia.*

A distinction is to be made between simple nervous dyspepsia (attended with pressure in the epigastrium, pain, vomiting, without hyperacidity) and acid dyspepsia (with marked hyperacidity, in addition to the foregoing symptoms). The nutrition is generally well preserved. The disorder occurs frequently in chlorotic and nervous individuals.

Diagnosis.—Ulcer is to be excluded. This is not always possible.

Treatment.—Cold sponging; active exercise; possibly iron; nutritious diet; massage; electricity; baths; psychotherapy.

D. Diseases of the Intestines.

13. *Intestinal Catarrh.*

Acute intestinal catarrh attends colds, infectious diseases, acute intoxications.

The more important *symptoms* are abdominal pains, colic, diarrhea, and tenesmus. The stools present a greenish appearance, from the presence of undecomposed bile, and there is considerable admixture of mucus, especially in cases of catarrh of the large bowel. Exhaustion may attend profound catarrhal states.

Treatment.—Rest in bed ; hot applications ; astringe
wines ; cocoa ; mucilaginous soups ; powdered opiu
(from 0.03 to 0.05—gr. ss–gr. ¾—frequently) ; opium a
tannic acid in powder ; tannigen (1.0—gr. xv), etc.

Chronic intestinal catarrh attends infectious diseas
alcoholism, diseases of kidneys and liver, etc.

Symptoms.—Alternating diarrhea and obstinate cons
pation for months or years. Sense of discomfort in t
abdomen, with flatulence and meteorism. The sto
contain abundant admixture of mucus. If the sm
intestine also be involved, the stools contain undigest
particles of food (lientery). When ulcerative proces
are present in the colon pus and blood appear in t
stools.

Membranous enteritis is attended with the painful d
charge of muco-membranous shreds, in conjunction wi
nervous intestinal catarrh and attacks of colic in neur
thenic individuals.

Treatment.—Courses at Carlsbad or Kissingen ; mil
diet ; warm packs ; massage ; systematic irrigation of t
bowels ; avoidance of indigestible and especially farin
ceous food, of fatty meats and of salads ; proscription
the use of alcohol.

In nursing infants *acute enteritis* due to spoiled mi
(infection) is characterized by diarrhea, with freque
greenish stools, often together with vomiting and loss
strength. Chronic diarrhea becomes attended with i
creasing cachexia (pedatrophy) in consequence of pr
gressive atrophy of the mucous membrane of the ent
gastro-intestinal tract.

Treatment.—Withdrawal of milk for several days, wi
the substitution of mucilaginous decoctions ; albumi
water ; artificial foods, hydrochloric acid, bismuth su
nitrate.

14. *Intestinal Tuberculosis.*

This occurs mostly as a complication in cases of p
monary tuberculosis (see p. 134).

Syphilis of the Bowel.—Syphilitic ulceration of t rectum is not at all uncommon, and recovery may be followed by stenotic cicatrices (funnel-shaped stenosis).

Symptoms.—Diarrhea, bloody stools, tenesmus, etc.

Treatment.—Antisyphilitic remedies; surgical dilatation for stenosis.

15. *Carcinoma of the Bowel.*

Carcinoma rarely develops in the small intestine. When it does, it appears in the form of solid tumors, usually freely movable and circumscribed, whose presence gives rise eventually to symptoms of occlusion of the bowel. The most common variety is carcinoma of the rectum, which may exist for a long time without occasioning symptoms, but which later causes chronic diarrhea, tenesmus, mucous and bloody stools, difficulty in defecation, sciatica, etc. Sometimes the progressive cachexia, or perhaps metastasis to the liver, may lead to digital examination of the rectum and the discovery of the new-growth.

The *treatment* must eventually be surgical.

16. *Stenosis and Occlusion of the Bowel* (Ileus).

Etiology.—Foreign bodies, fecal accumulations, tumors, cicatricial strictures, axial torsion, incarcerated hernia, invagination (in children), constriction of the bowel (internal hernia, abnormal ligaments or bands).

Stenosis is attended with difficulty in defecation (the stools being band-like or globular), abdominal pains, meteorism, nausea.

Occlusion is attended with irremediable constipation, together with cessation of flatulent discharge, severe nausea and retching, and abdominal pain, vomiting, at first bilious, later feculent, constant eructation, hiccough, and marked prostration.

Diagnosis.—Inquiry should be made as to the cause of the condition and the usual sites of hernia examined. Intussusception is attended with mucous and bloody stools, and the presence of a sausage-shaped tumor.

Treatment.—Immediate celiotomy or herniotomy; opium (0.05—gr. ¾—every two hours); ice; lavage of the stomach; rectal irrigation.

17. *Habitual Constipation.*

This occurs in nervous, hypochondriacal, and a
individuals, and especially in children and wome
occurs in conjunction with chronic catarrhal states
stomach and the intestines, and with sedentary o
tions.

Treatment.—Vegetables and fruits in abundance
bread, bran-bread ; active exercise in the open air ;
age of the abdomen ; frictions ; enemata of glycer
oil, and of water ; faradization ; courses at Kiss
Carlsbad, Franzensbad, etc. ; possibly rhubarb, c
sagrada, tamarinds, aperient waters.

When *hemorrhoids* are present : cold affusions ;
vision for regular evacuation of the bowels ; hemorr
pessary ; radical operation.

18. *Intestinal Parasites.*

1. *Tape-worms* (see p. 128) occasion unpleasant
tions, digestive derangement, nervous states, diarrh

In a suspected case castor-oil should be given an
stools examined for proglottides and ova.

Treatment.—Extract of filix mas (2.0—gr. xx
capsules, five or six at intervals of half an hour), fol
in three hours by a tablespoonful of castor-oil ever
hours. Particular care should be taken to secure the

2. *Ascarides* (see p. 126).—Santonin (0.05—grai
several times daily).

3. *Oxyures* (see p. 126) cause annoying itching
region of the anus. The treatment consists in th
ministration of enemata of garlic rubbed up in
naphthalin (0.15 to 0.4—gr. ijss–gr. vj—four tin
twenty-four hours); castor-oil ; calomel.

4. *Ankylostomum* (see p. 126) requires the same
ment as tape-worm ; drastics.

E. Diseases of the Peritoneum.

19. *Perityphlitis.*

This is a form of local peritonitis following perfo
of the vermiform appendix, which usually results i

sequence of fecal accumulation in the appendix (fecal concretions).

Symptoms.—Rather sudden occurrence of abdominal pain, especially in the right iliac fossa, with vomiting, eructation, constipation, meteorism, and moderate fever.

There is dulness on percussion in the ileo-cecal region (right iliac fossa), with a sense of resistance, due to exudation and adhesive peritonitis. If the inflammation of the peritoneum be progressive and attended with accumulation of pus in the abdominal cavity, the fever, which had previously subsided, rises higher than it had been, while the pulse becomes softer and more frequent, the meteorism more marked, and the general condition more grave, and hiccough and collapse appear.

Treatment.—Tincture of opium, 20 drops every two hours, until complete relief from pain is secured; ice-bag; cold drinks; no purgatives. Enemata may be employed after the disease has pursued a favorable course for a week. If aggravation takes place, as well as when an abscess forms, immediate celiotomy is to be undertaken (exploratory puncture is entirely without danger).

20. *Acute Peritonitis.*

This usually occurs in conjunction with rupture of abscesses (perinephric, parametric, subphrenic, hepatic, tubal), with ulcerations of stomach and bowels (gastric ulcer, duodenal ulcer, typhoid fever, dysentery, intestinal tuberculosis), with incarceration of the bowel; further in cases of pyemia and of puerperal fever and in the sequence of pleurisy and pericarditis, and as a result of injuries.

A distinction is to be made between *circumscribed peritonitis* (rapid formation of adhesions, without constitutional infection) and *diffuse peritonitis;* further between *sero-fibrinous* and *purulent* and *putrid peritonitis* (dependent upon infectious agents: toxins, staphylococci, streptococci, bacterium coli commune, proteus, etc.).

Symptoms.—Severe abdominal pain, vomiting, greatly distended abdomen, elevation of the diaphragm. The

percussion-note is tympanitic everywhere ; or if
tion have taken place, there is dulness in the dep
portions. When air is present within the abdominal
(perforative peritonitis, see p. 96) the liver-dulnes
when the patient lies upon the right side the spleni
ness, disappears through displacement by the air.
the lapse of several hours there is rapid failure
patient, who presents a collapsed appearance (H
cratic facies), with heart-failure.

Treatment.—Circumscribed peritonitis is to be t
with opium, etc. (see perityphlitis) ; diffuse perit
(after exploratory puncture) by celiotomy, ice, opium
0.05 to 0.1—gr. ¾–gr. jss).

21. *Chronic Peritonitis.*

This is usually tuberculous (see p. 136). A disti
is to be made between a *dry* (adhesive) and an *exu*
variety. The former leads to extensive adhesions
intestine, with unfavorable prognosis and marasmus ;
the latter recovery may take place after celiotomy
vided the operation be not undertaken too late.

Among the *symptoms* are persistent fever, abdo
pain, presence of a serous exudate (as demonstrate
exploratory puncture) and of tuberculosis elsewhere

Peritoneal tuberculosis is frequently attended wit
presence of large tuberculous tumors of the ome
and hemorrhagic exudations.

The *prognosis* is unfavorable.

22. *Ascites.*

This consists in an accumulation of fluid in the p
neal cavity in consequence of venous stasis. It occ
conjunction with diseases of the liver and portal
(cirrhosis, carcinoma, thrombosis), with general v
stasis (heart-disease in the stage of derangement of
pensation), and with diseases of the kidneys.

Among the *symptoms* are distention of the abd
with especial prominence of the lateral portions ;
tion of the diaphragm ; dulness on percussion i
dependent portions ; with a sense of fluctuation.

dulness varies with change in posture, as the effusion usually finds its way to the most dependent part. Frequently there is edema of the lower extremities, from pressure upon the ascending vena cava. Exploratory puncture discloses the presence of a serous exudate, the fluid exhibiting a specific gravity below 1015 (see p. 95). The differential diagnosis from ovarian cysts, etc., is discussed on p. 94 *et seq.* In these other conditions there is wanting especially the change in percussion-note with change in posture, etc.

Treatment should be directed to the primary disorder; digitalis; potassium and sodium borotartrate; calomel; aspiration.

23. *Carcinoma of the Peritoneum.*

This usually occurs by metastasis from stomach, liver, rectum, or uterus. It is attended with a copious ascitic effusion, usually hemorrhagic, with tumors of the omentum, pain, cachexia.

F. Diseases of the Liver.

24. *Catarrhal Jaundice.*

This results from extension of catarrhal swelling from the duodenum to the biliary passages, and may be of infectious character. The obstruction to the flow of bile into the intestine leads to the development of jaundice, together with anorexia, nausea, vomiting, abdominal pain. The jaundice may last from two or four to eight weeks. It is characterized by a yellowish discoloration of the skin of the whole body and of the sclera, together with annoying itching, slowing of the pulse, and gastric derangement. The stools are clay-colored, from the absence of biliary coloring-matter and the presence of considerable unabsorbed fat and fat-needles. The urine is brown, and on shaking forms a yellow foam (see p. 112). The liver may be enlarged from stasis of bile and the gall-bladder may be dilated.

The *prognosis* is favorable.

Treatment.—Carlsbad, Mühlbrunnen, Kissingen waters, etc.; diet; no fat.

Chronic jaundice results in rare cases as a conseq of obliteration of the choledoch duct (chronic f cholangitis). It is attended with progressive emaci cachexia, convulsions, coma, delirium, hemorr (cholemia, intoxication).

Weil's disease is probably an infectious dis attended with fever, jaundice, herpes, rheumatic enlargement of the spleen, and nephritis. It las two weeks, and the prognosis is good.

25. *Cholelithiasis.*

The formation of calculi (cholesterin, bilirubin, salts) in the biliary passages and in the gall-bl occurs most commonly in women between 40 a years of age. Hereditary predisposition appears to influence in the etiology.

The principal *symptom* is biliary colic, dependent impaction of a calculus in the biliary passages. consists in intense pain in the region of the liver an stomach, with vomiting, chills, fever, enlargement o spleen. Such attacks of colic, lasting from one o to four days, may be repeated at intervals of va length. At the conclusion of the attack jaundice ap if the stone have not been impacted in the cystic du which situation it is capable of giving rise only to in the gall-bladder (dropsy of the gall-bladder), th absorption of the bile when the case pursues a ch course). Permanent impaction gives rise to chronic dice. As a result of ulceration, abscesses may for consequence of migration of cocci from the bowel, the occurrence of pyemic chills, perforative perit metastatic suppurative processes, etc.

If the gall-bladder be filled with calculi, these m palpable, with the development of crepitation. stones may be present in the stools.

Diagnosis.—Colic associated with gastric ulcer an vous attacks unattended with jaundice are to be excl

The *prognosis* is dubious. Frequent repetition unfavorable; as is also chronic jaundice.

Treatment.—In the attacks, poultices, extract of opium (0.06 to 0.1—gr. j-gr. jss—several times daily), morphin subcutaneously; later, enemata; then a course at Carlsbad; sodium salicylate, sodium bicarbonate, of each 15., a small quantity thrice daily.

26. *Cirrhosis of the Liver.*

A distinction should be made between *hypertrophic* and *atrophic cirrhosis*, the latter representing frequently the terminal stage. The affection occurs most commonly in conjunction with chronic alcoholism, also with tuberculous peritonitis and chronic malaria.

In the *first stage* the liver is enlarged, through infiltration and fatty degeneration. Its surface is hard, its border smooth. Slight jaundice is common. The spleen is enlarged and chronic gastrointestinal catarrh is present.

In the *second stage* the liver is diminished in size, from disappearance of parenchyma and hyperplasia of the interstitial connective tissue. The surface of the organ is nodular and hard, its margin irregular. Jaundice may be wanting.

The occurrence of stasis in the portal vein is manifested by ascites, swelling of the spleen, chronic gastrointestinal catarrh, distention of the abdominal veins, and progressive cachexia.

Complications.—There are frequently also contracted kidney, hypertrophy of the heart (alcohol), etc.

Treatment.—Course at Carlsbad; calomel; diuretics; digitalis; aspiration; prophylaxis.

27. *Acute Yellow Atrophy of the Liver.*

This is a profound infectious disease terminating in death in the course of a few days.

It sets in with vomiting, fever, and jaundice. Consciousness is greatly disturbed (coma and delirium). The liver becomes smaller from day to day in consequence of fatty degeneration and resorption. The urine contains leucin, tyrosin (see Plate 15, Figs. 3 and 4), and much ammonia, with diminished elimination of urea.

28. *Carcinoma of the Liver.*

This is usually metastatic from stomach, rect
esophagus; less commonly primary in the biliary pa
It gives rise to the presence of irregular enlargem
the liver, often to an enormous degree, together w
presence of multiple palpable tumors. There is
jaundice, more rarely ascites. Progressive cache
feature.

29. *Syphilis of the Liver.*

Tertiary syphilis may be attended with the de
ment of gummata in the liver, with infiltration and
faction, secondary cicatricial constriction, giving
lobulation. Ascites and enlargement of the sple
common, jaundice rare. Pain is a marked sympto
marasmus may ensue.

Treatment.—Potassium iodid and mercurial inun
not always successful.

30. *Abscess of the Liver.*

This occurs in conjunction with cholelithiasis, p
ulceration of the bowel, etc. It is characterized
occurrence of severe chills, with high fever, attac
intense pain, vomiting, enlargement of the liver,
jaundice, no ascites, eventually fluctuation (explo
puncture).

The *prognosis* is unfavorable. Rupture may take
and excite perforative peritonitis.

Treatment.—Surgical intervention.

31. *Echinococcus* (see p. 128) *of the liver* give
to enlargement of the organ, with the developme
fluctuating, tensely elastic tumors. Hydatid fr
(audible vibration on shock-like tapping) is often p
Eventually there may be also ascites, enlargement
spleen, jaundice. Exploratory puncture disclos
presence of fluid free from albumin, and containing
succinic acid and sodium chlorid (see Plate 21, F
together with hooklets, scolices, and portions of
brane (see Plate 21, Fig. 8).

Treatment.—Surgical intervention.

32. *Cyanotic (Nutmeg) Liver.*

This condition of the liver occurs in conjunction with all forms of heart-disease in the stage of derangement of compensation. There is pain on pressure in the region of the liver and the abdomen is prominent. The surface of the liver is hard, its margin sharp and smooth. At the same time there is usually ascites, the liver remaining large after its disappearance. The urine is scanty and concentrated, and edema, cyanosis, etc., are likely to be present.

Treatment.—Digitalis; diuretics; aspiration of the ascites.

Fatty degeneration of the liver occurs in conjunction with anemia, phosphorus-poisoning, arsenical poisoning, excessive indulgence in alcohol (see cirrhosis), and cyanotic liver.

The condition is mostly unattended with distinctive symptoms. The liver is usually large and its margin smooth. At times secondary atrophy follows.

Amyloid degeneration of the liver occurs in conjunction with chronic suppurative processes, tertiary syphilis, chronic pulmonary tuberculosis, caries, etc. The liver is large, hard, and dense, with a blunt margin. There are usually at the same time enlargement of the spleen and amyloid degeneration of the kidneys.

33. *Constricted Liver.*

This deformity results generally from the long-continued use of constrictions about the waist, and it is accordingly more common in women than in men. It can often be detected by palpation beneath the right half of the costal arch. Sometimes a distinct fissure can be felt. The condition is attended with pain, and predisposes to the formation of biliary calculi.

34. *Thrombosis and Inflammation of the Portal Vein.*

Thrombosis of the portal vein occurs as a result of compression by tumors (adjacent carcinoma, syphilis), of cicatricial contraction (cirrhosis, etc.). It gives rise in turn to ascites, enlargement of the spleen, gastric and intestinal hemorrhages, caput Medusæ (dilatation of the

veins surrounding the umbilicus, with the format
anastomoses). If agents capable of inducing infl
tion gain access to the thrombus (in conjunctio
ulceration of the stomach, or bowels, gall-stones, pe
litic abscesses, inflammation of the umbilical v
infants, etc.), symptoms of profound pyemia deve
addition to those enumerated (see abscess of the li

Diagnostic Features of the more Common Diseases
Liver.

	Volume.	Consistence.	Liver-margin, liver-surface.	Jaundice.	As sple larg
Cyanotic Liver	Enlarged.	Firm.	Smooth.	Absent.	Pres
Acute Yellow Atrophy	Diminished.	Firm.	Smooth.	Absent.	Abse
Cirrhosis (First Stage)	Enlarged.	Firm.	Smooth.	Absent.	Abse
Cirrhosis (Second Stage)	Diminished.	Hard.	Nodular.	Present.	Pres
Cholelithiasis	Enlarged.	Firm.	Smooth; possibly distention of gall-bladder.	Present.	Abse
Carcinoma	Enlarged.	Hard.	Nodular	Present.	Pres
Syphilis	Enlarged.	Hard.	Lobulated.	Present.	Pres
Echinococcus	Enlarged.	Soft.	Lobulated.	Absent.	Abse
Amyloid Disease	Enlarged.	Hard.	Blunt.	Absent.	Abse
Abscess	Enlarged.	Soft.	Bulging.	Absent.	Abse

G. Diseases of Spleen.

35. *Enlargement of the Spleen.*

This may occur as the result of stasis (cyanotic s
in conjunction with diseases of the liver and the
further with infectious diseases (typhoid fever, m
fever, pyemia, etc.), with embolism (endocarditis, a
sclerosis), with amyloid disease (see Amyloid Liv
203), with leukemia (p. 221). Symptoms arise
when the enlargement is extreme (leukemia). The

marked are painful sensations, especially upon muscular effort, and dyspnea.

In all of the conditions named the spleen is distinctly palpable as a hard, solid tumor. Frequently a depression (incisure) can be felt in the body of the organ. A *wandering spleen* also may become palpable.

The *treatment* varies in accordance with the primary disorder, and may require the administration of arsenic or quinin, or the application of bandages.

H. Diseases of the Pancreas.

Of these may be mentioned atrophic processes occurring in some cases of diabetes, hemorrhages (sometimes leading to sudden death from anemia), carcinoma, and cystic formations (see Plate 61).

The *diagnosis* may require exploratory puncture, and the *treatment* surgical intervention.

V. DISEASES OF THE UROPOIETIC ORGANS.

A. Diseases of the Kidneys.

1. *Acute Hemorrhagic Nephritis.*

The anatomic alterations consist in cloudy swelling, fatty degeneration, and necrosis of the renal epithelium.

The disease is less commonly primary (from exposure to cold), but it occurs usually in conjunction with acute infectious diseases (scarlet fever, diphtheria, small-pox, endocarditis, etc.) as a result of intoxication. It may also occur in consequence of poisoning with cantharides, potassium chlorate, turpentine, salol, etc. The duration of the disease is from three or six to ten weeks.

The *symptoms* include headache of gradually increasing severity, nausea, vomiting, edema of the eyelids, the face, the extremities, the scrotum, ascites, hydrothorax, hydropericardium.

This form of renal dropsy depends probably upon an abnormal permeability of the walls of the vessels induced by the activity of toxic metabolic processes, and n result of stasis (see Cardiac Dropsy, p. 61).

The *urine* is much diminished in amount and is c trated. It presents the appearance of blood and tains much albumin. The abundant sediment cc many hyaline and epithelial tube-casts, white an blood-corpuscles and renal epithelium, while the ur chlorids, and the phosphates are diminished. The s gravity is high.

Through the retention of the substances name gether with others (extractives), there results ur coma, somnolence, epileptiform and eclamptic at vomiting, headache, amaurosis. In reaching a *dia* chronic recurring nephritis is to be excluded.

The *prognosis* is rendered grave by the existen effusions into the serous cavities (hydrothorax, etc by uremia. The disease may be gradually transf into chronic nephritis.

Treatment.—Milk-diet, hot packs, diaphoresis baths, diaphoretics (tea, pilocarpin, 0.02 [gr. ¼]), talis, mild diuretics (sodium acetate, theobromin salicylate), aspiration. If uremia develop, enema digitalis may be administered, together with mor

2. *Chronic Nephritis.*

Subchronic and chronic nephritis may develop i sequence of acute nephritis, in conjunction with al ism and in many cases without obvious cause.

Chronic parenchymatous nephritis, occurring as a ary disorder, with fatty degeneration of the renal e lium, is present in many cases unnoticed for a long until increasing emaciation and anemia, disturban the activity of the heart, chronic catarrhal cond (from stasis), the occurrence of edema or uremia respiratory difficulty and migraine) cause the pati seek professional advice.

Chronic nephritis causes almost always disturban the nutrition and in blood-formation, thus leading t ondary anemia. Hypertrophy of the heart also ma place (see Contracted Kidney, p. 213). Uremia and edema are of common occurrence.

In addition to the acute variety of uremia, it is the chronic variety, characterized by migrainous conditions, headache, vomiting, diarrhea, dyspnea (uremic asthma), that commonly occurs. There may be also albuminuric retinitis. The *urine* is usually secreted in diminished amount. It is usually turbid and deposits considerable sediment. There may be considerable admixture of blood and a large amount of albumin. The disease lasts from one or two to four years, and recovery is uncommon.

The following varieties of chronic nephritis may be distinguished:

(*a*) Large red, variegated kidney; chronic hemorrhagic nephritis. The sediment contains many tube-casts, together with white and especially red blood-corpuscles.

(*b*) Large white fatty kidney, with fatty degeneration of the renal epithelium. The sediment contains tube-casts covered with large numbers of fat-globules, and much fattily degenerated renal epithelium and leukocytes.

(*c*) Secondarily contracted kidney (connective-tissue contraction). This is frequently the terminal stage of the foregoing varieties, if uremia, edema, or heart-failure have not previously led to a fatal termination. In this form of the disease the amount of *urine* secreted is more abundant than in the forms described under (*a*) and (*b*), while the secretion itself is of light color and the sediment smaller; admixture of blood is uncommon. Hypertrophy of the heart develops as a rule.

The *diagnosis* is considered on p. 215.

Treatment.—Milk-diet; warm baths; cold frictions; abstinence from all irritating articles of food (condiments, alcohol); Faching, Wildung, and Bilin waters; avoidance of cold; iron; iodin; arsenic.

3. *True Contracted Kidney.*

Granular atrophy of the kidney (increased development of the interstitial connective tissue, with contraction, and destruction of the renal parenchyma) develops in conjunction with chronic alcoholism, arteriosclerosis, gout, lead-poisoning; less commonly with syphilis, malaria, etc. The duration of the disease is from four or six to ten or more years. In consequence of the retention of certain noxious metabolic products there results contraction of the peripheral small arteries, giving rise to a tense pulse, and in consequence to increase in the arterial pressure. The circulatory obstruction thus occasioned is neutralized by hypertrophy of the left ventricle (compensation). For a time the increased work of the heart is adequate for the needs of the circulation, but finally derangements of compensation set in (catarrhal states of the bronchi, the stomach, the intestines, etc., from stasis, respiratory difficulty, pneumonia). There may now appear also edema and ascites (in consequence of stasis in the circulation). Frequently there are marked anemia and albuminuric retinitis.

The *urine* is secreted in large amounts (from 3000 to 4000 cu. cm.). It is pale, and its degree of concentration slight. The specific gravity is quite low, and there is marked diminution in all the solid constituents (urea, etc.). The amount of albumin present is small and at times it may be entirely wanting. The sediment is scanty, and only individual tube-casts and white blood-corpuscles, as well as epithelial cells, can be found.

The *diagnosis* is indicated in the table on p. 215.

Treatment.—Removal of the cause of the disease (alcohol, lead); administration of alkaline waters; employment of baths (Nauheim, Baden-Baden, Kissingen, iron springs, etc.); observance of a milk-diet, etc.; digitalis; potassium iodid.

4. *Cyanotic Kidney.*

This condition occurs in conjunction with diseases of the heart (derangements of compensation) and venous hyperemia. The retarded circulation of blood through the kidneys exerts an injurious influence upon the renal epithelium.

The *urine* is much diminished in amount, greatly concentrated, and dark in appearance. It contains a moderate amount of albumin and is of high specific gravity. The sediment is scanty and contains hyaline tube-casts and a small number of white and red blood-corpuscles.

Treatment.—Digitalis.

Hemorrhagic infarction also occurs in conjunction with disease of the heart (endocarditis). Symptoms are often wanting, and they may appear as transitory slight hematuria, or pains in the loins (perinephritis).

5. *Amyloid Kidney.*

For the *etiology*, see Amyloid Liver, p. 209.

The *urine* is usually abundant, of pale-yellow color, and of normal or diminished specific gravity. It contains a large amount of albumin and deposits a slight sediment consisting of isolated white blood-corpuscles and hyaline tube-casts. Hypertrophy of the heart does not take place. Frequently marked edema (amyloid disease of the vessels?) occurs, while uremia and retinitis do not. Eventually progressive anemia and marasmus make their appearance.

Diagnosis of Diseases of the Kidney.

	Acute Nephritis.	Chronic Parenchymatous Nephritis.	True Contracted Kidney	Cyanotic Kidney.	Amyloid Kidney.
Amount of Urine	Small.	Normal.	Very abundant.	Very small.	Abundant.
Specific Gravity	High.	Normal.	Low.	High.	Low.
Amount of Albumin	Abundant.	Abundant.	Small or none.	Small.	Abundant.
Sediment	Much.	Much.	Little.	Little.	Little.
Hypertrophy of the Heart	Absent.	Usually present.	Always present.	Generally present.	Absent.

6. *Pyelonephritis.*

Catarrhal and suppurative inflammation of the pelvis of the kidney, with or without suppurative inflammation of the kidney, is usually secondary to general infection

(pyemia, perforative peritonitis, etc.), renal calcul
itis (ascending), and especially when paralysis
bladder exists, in the puerperal state, etc.

If the disorder be of metastatic origin, embolic
tions of bacteria (staphylococci, streptococci) can be
in the substance of the kidney. The most im
excitant of ascending pyelitis, other than those
named, is the bacterium coli commune.

Symptoms.—Severe pain in the loins and in the
of the ureter; irregular fever, with chills, sweats
tration; abundant urinary sediment consisting in p
pure pus, in part of admixture with epithelium fro
pelvis of the kidney and red blood-corpuscles (ca
less commonly tube-casts (toxic nephritis). The u
usually of acid reaction. In the event of perin
abscess-formation there is increase in the pain,
edema of the adjacent soft tissues.

Diagnosis.—Tuberculosis is to be excluded, by
ination of the urinary sediment for tubercle-bacilli.
etiologic factor is to be looked for.

Treatment.—Attention should be directed to the
ary disorder; milk-diet; Wildung, Vichy, Neu
waters; balsams (copaiba, turpentine), astringents
nic acid, lead acetate) internally; also salicylic aci

Eventually surgical treatment may be required.

7. *Nephrolithiasis.*

Excretion of renal sand, constituted principal
phosphates or urates (see p. 114), often takes place,
out distinctive symptoms. If, however, large c
tions form in the pelvis of the kidney, impaction
the ureter may take place and give rise to attacks of
colic. These are characterized by pain of the utm
tensity, radiating from the region of the kidney t
the groin and the scrotum, together with anuria, col
and vomiting. Such attacks may last from several
to days.

In consequence of the mechanical irritation
results catarrhal, perhaps ulcerative, pyelitis, wh

characterized by the presence of pus and especially of blood in the urine. The disorder usually pursues a chronic course, and it may be complicated by the formation of abscesses and of purulent pyelonephritis.

If the urine be acid, uratic calculi, if alkaline phosphatic calculi especially form.

Diagnosis.—Of importance are the character of the attacks of colic and the occurrence of hematuria.

Treatment.—Moderation in the use of meat, with a preponderance of vegetable food; abstinence from alcohol; abundance of liquids, especially the alkaline waters; active bodily exercise, systematic gymnastics; sodium bicarbonate, lithium bicarbonate, urea (10.—2½ drams—daily), lysidin, piperazin, etc.

If the calculus be constituted of phosphates, acids, lactic acid (from 0.5 to 1.—gr. vijss–gr. xv—in solution) or salicylic acid, may be administered.

In attacks of colic opium, morphin, hot packs, and cutaneous irritants may be employed.

Surgical intervention may be ultimately required.

8. *Tuberculosis of the Kidney.*

Eruptions of tubercles in the kidney, with caseation in the structure of the organ and in the pelvis of the kidney, occur usually in conjunction with tuberculosis in other portions of the body (lungs, glands, prostate, bladder, etc.).

Among the *symptoms* are those of purulent pyelonephritis, without indications of pyemia, together with abundant elimination of pus and the presence of caseous matters and of tubercle-bacilli in the urine.

Treatment.—If the disease be unilateral, as determined by the use of the cystoscope, the affected kidney should be extirpated. Other measures to be employed are mentioned under pyelonephritis.

9. *Tumors of the Kidney* (see p. 99).

10. *Hydronephrosis.*

Accumulation of fluid within the pelvis of the kidney occurs in consequence of kinking or obstruction of the ureter (renal calculi, tumors, wandering kidney, pregnancy, inflammatory processes, tumors of the prostate, tumors of the bladder), and of atrophy of the kidney from pressure.

The swelling that results is of variable size in consequence of the varying amount of fluid present (in contradistinction from echinococcus), and urea can be demonstrated in the fluid obtained on exploratory puncture, although this may be wanting if the disorder have existed for a long time. The diagnosis is considered upon p. 99.

11. *Wandering Kidney.*

This condition occurs frequently in women, and by reason of the changes in the position of the organ gives rise to pain, with especial frequency upon the right side, in the sacral region, and the abdomen. It is attended further with vomiting, attacks of colic, and hydronephrosis if the ureter becomes kinked.

The *diagnosis* is based upon the presence of a movable, sensitive, kidney-shaped body in one or other hypochondrium, as determined by bimanual examination, while the normal area of renal dulness is wanting.

Treatment.—Massage; compressive and supporting bandages; finally nephrorrhaphy.

B. Diseases of the Bladder.

12. *Cystitis.*

Acute catarrh of the bladder occurs in conjunction with infectious diseases (typhoid fever, etc.), as a result of catheterization, in conjunction with intoxications, with pregnancy, and in the sequence of gonorrhea, etc.

The *symptoms* consist in pain in evacuation of the urine, frequent desire to urinate, spasm of the sphincter vesicæ, dull pain in the region of the bladder and fever.

The *urine* is turbid and contains much mucus, as well as blood, epithelial cells, and pus-corpuscles. On standing, it speedily undergoes ammoniacal fermentation, with the development of coffinlid and thorn-apple crystals (see Plate 18, Fig. 1).

Bacteria also are present in large numbers.

Chronic catarrh of the bladder results especially in conjunction with paralysis of the bladder (attending diseases of the spinal cord), with strictures of the urethra, stone in the bladder, and hypertrophy of the prostate.

Diagnosis.—Inquiry should be directed toward the etiologic factor. The presence of stone in the bladder is indicated by repeated hematuria and pain radiating toward the head of the penis, with at times retention of urine. Examination with the sound may confirm the diagnosis. Tuberculosis is to be excluded.

Treatment.—Removal of vesical calculi; treatment of strictures; abundant use of liquids (much milk, infusion of uva ursi, Wildung and Faching waters); no beer, etc.; hot cataplasms, morphin-suppositories; belladonna.

In the more chronic varieties of the disorder balsam of copaiba, balsam of tolu, turpentine, tannic acid, irrigation of the bladder with solutions of boric acid or zinc sulphate, or with emulsion of iodoform, will prove useful.

Tuberculous Cystitis.—The symptoms and conditions are much the same as those that have just been described, together with abundant elimination of pus and the presence of tubercle-bacilli in the urine.

Treatment.—In addition to that indicated, surgical intervention may be necessary.

13. *Tumors of the Bladder.*

These are usually papillomata (improperly designated villous carcinomata), less commonly true carcinomata.

The *symptoms* include recurring hematuria and pain, together with evidences of chronic cystitis.

The *diagnosis* is to be determined by palpation through the rectum or the vagina, through catheterization and the cystoscope.

The *treatment* may finally require surgical intervention.

14. *Nocturnal Enuresis.*

This disorder is observed in conjunction with nocturnal epilepsy and in nervous, debilitated children, from weakness of the sphincter vesicæ.

The *treatment* includes cold sponging, regularity in evacuation of the urine, electricity, elevation of the pelvis by raising the foot of the bed, etc.

VI. DISEASES OF THE BLOOD, AND OF THE BODILY METABOLISM.

A. Diseases of the Blood (Plates 1 to 4).

1. *Chlorosis.*

This occurs commonly in girls at the period of puberty. It is attended with derangement in blood-formation and in gastro-intestinal digestion, and with nervous disturbances. There is pallor of skin and mucous membranes, with frequent gastric disturbances, vomiting, headache, lassitude, drowsiness, palpitation of the heart (accidental murmurs), venous hum, shortness of breath (from deficiency of oxyhemoglobin).

Examination of the *blood* discloses no special diminution in the number of erythrocytes, but, on the contrary, marked deficiency in hemoglobin (for method of determination, see p. 75). The number of leukocytes remains normal.

Complications.—Diseases of the myocardium (dilatation), ulcer of the stomach, hysteria.

Diagnosis.—Tuberculosis is to be excluded. Emaciation is usually wanting.

Treatment.—Rest; sleep; milk; eggs; cool frictions; salt baths; iron (Blaud's pill, ferratin, etc.). The administration of alcohol (in the form of red wine) is quite without utility. Steel baths may be advised.

2. *Anemia.*

Acute anemia attends marked loss of blood occurring in consequence of injuries of arteries, menorrhagia, etc. Among the *symptoms* are pallor of the face and an appearance of collapse, coldness of the skin, a condition of coma, convulsions, amaurosis (anemia of the retina).

Treatment.—Control of hemorrhage by means of tampons; depression of the head; provisional bandaging of the extremities; saline infusions; injections of camphor and ether; enemata of water.

Chronic secondary anemia is the common sequel of all chronic wasting diseases, especially of chronic nephritis, tuberculosis, carcinoma, chronic suppuration, gastric and intestinal catarrh, malaria, chronic intoxications (lead, mercury, etc.). It attends also ankylostomiasis, the presence of teniæ, and it occurs in cases of neurasthenia, etc.

Symptoms.—Lassitude, anorexia, sense of fear, headache, palpitation of the heart, pallor, gastric derangement, and a host of other nervous disturbances.

Examination of the *blood* shows the number of red corpuscles to be greatly diminished, with changes in form and size (poikilocytes, microcytes, macrocytes). Nucleated red corpuscles are uncommon. The leukocytes are frequently increased in number, but only the polynuclear variety. The proportion of hemoglobin is diminished in correspondence with the number of red corpuscles.

The *prognosis* is dependent upon the nature of the primary disorder.

Treatment.—Rest; baths; forced feeding; massage; electricity; iron; arsenic; sojourn in the mountains.

Pernicious anemia (primary anemia) is progressive in course and almost always terminates fatally. It is attended with hemorrhages into the retina, petechiæ, and fatty degeneration of various organs and structures. There is constant diminution in the number of erythrocytes, to as low as 400,000 in the cu. mm., and nucleated red blood-corpuscles are usually present (normoblasts and gigantoblasts). The proportion of hemoglobin is reduced, but usually not in correspondence with the oligocythemia. The number of leukocytes remains normal.

The *etiology* of the disorder is obscure.

The *treatment* does not differ materially from that of secondary anemia. Arsenic is the most reliable remedy. Bone-marrow has been used.

3. *Leukemia* (Plates 4 and 60).

This disorder is probably dependent upon disease of the blood-forming organs (the spleen, the bone-marrow, the lymphatic glands). It is characterized by diminution in the number of erythrocytes, with increase in the number of leukocytes, and is manifested clinically by progressive anemia and cachéxia. The disease may last from one to two years, and its cause is quite unknown. A distinction is made between lymphatic, myelogenous, and splenic varieties, but as a rule the type of the disease is mixed. The first is characterized especially by great enlargement of the lymphatic glands, the last by marked increase in the size of the spleen.

The disorder is gradual in onset and attended with progressive emaciation, anemia, respiratory difficulty, and pain in the side. The abdomen becomes swollen, and there is frequently irregular fever, with nervous disturbances and pain in the bones, especially on percussion, and particularly over the sternum.

In addition to the enlargement of the spleen, which may reach enormous proportions, there are frequently increase in the size of the liver (from infiltration) and also infiltration of the kidneys and of the retina, and hemorrhages into the skin.

The *blood* presents a yeast-like color. The number of erythrocytes is greatly diminished (to as low as 2,500,000 or even 1,000,000 and less), and frequently nucleated red blood-corpuscles are present. The proportion of hemoglobin also is diminished. The number of leukocytes is enormously increased (up to from 200,000 or 400,000 to 600,000 in the cu. mm.). The proportion of red to white corpuscles may thus reach 2 to 1. The increase in the leukocytes involves less the polynuclear (see leukocytosis, p. 76) than the mononuclear cells. Especially numerous are found medullary cells (myelocytes), which are usually not present in normal blood. Besides, the eosinophile cells also are often increased (see Plate 4).

In cases of *lymphemia*, the small mononuclear cells (associated with enlargement of the lymphatic glands) preponderate; in cases of the more common, *splenomed-*

ullary, variety, the eosinophile and the ~~medullary~~ cells; in cases of *acute leukemia* (terminating fatally in the course of several weeks), also the mononuclear cells (usually in conjunction with a pronounced hemorrhagic diathesis). In the bodies of subjects dead of leukemia Charcot-Leyden crystals form in the blood. In the urine the uric acid is usually increased.

Treatment.—Arsenic, quinin, potassium iodid, iron iodid, phosphorus, richly albuminous diet, baths, etc.

Pseudoleukemia is attended with the presence of symptoms similar to those of leukemia (splenic enlargement, cachexia, anemia), but without increase in the number of leukocytes.

Hodgkin's disease is characterized by the presence of multiple glandular enlargement (lymphosarcoma). It pursues a rapidly pernicious course.

The state of the *blood* appears to be normal.

The *treatment* conforms to that just indicated.

4. *Hemoglobinemia.*

Destruction of red blood-corpuscles in the vessels, with the presence of free hemoglobin in the blood, occurs in conjunction with various forms of poisoning (potassium chlorate, anilin, nitrobenzol, antifebrin, hydrogen arsenite, toadstools, etc.), in the sequence of transfusion and of ~~burns~~, and in conjunction with profound infection (syphilis, scarlet fever, etc.).

Symptoms.—Chills, fever, vomiting, languor, slight jaundice.

The *urine* contains hemoglobin (see p. 111), without the presence of red blood-corpuscles in the sediment.

The *prognosis* is usually favorable.

5. *Hemorrhagic Diathesis.*

A tendency to the extravasation of blood under the skin (petechiæ), to bleeding from the gums, to hemorrhage from the bowels and beneath the periosteum, occurs in conjunction with various diseases.

The *etiology* is unknown.

(*a*) *Scorbutus* occurs frequently in sailors, from want of fresh vegetables, of variation in diet, and as a result of exposure to cold, etc. Amid symptoms of general prostration there occur numerous hemorrhages beneath the skin and mucous membranes, as well as from the stomach, the kidneys, etc. There may be, further, ulcerative stomatitis, swelling of the joints, etc.

Treatment.—Vegetables, fruit, lemonade, quinin, etc.

(*b*) *Purpura* (morbus maculosus Werlhofii) is characterized by multiple hemorrhages into the skin, by fever, and by prostration.

(*c*) *Peliosis rheumatica* is attended with swelling of the joints and pain, with hemorrhages, especially in the extremities.

Treatment.—Ergotin, quinin, salicylic acid.

(*d*) *Hemophilia* (bleeders' disease) consists in a familial tendency to severe hemorrhages, even upon the slightest provocation, as from cuts, extraction of teeth, etc.

B. Disorders of the Bodily Metabolism.

6. *Diabetes Mellitus.*

The designation *glycosuria* is applied to the transitory elimination of glucose in the urine. This occurs in the sequence of various forms of poisoning (hydrocyanic acid, morphin, phloridzin), of traumatism, especially of the nervous system, concussion of the brain, etc. (puncture-center of the medulla oblongata).

The designation *diabetes mellitus*, on the other hand, is applied to the inability of the body to consume the sugar taken up from the gastro-intestinal tract, together with the persistent elimination of this substance in the urine. Several varieties of the disorder are distinguished:

Alimentary Glycosuria.—Elimination of glucose only after ingestion of food containing sugar; this is a mild form.

Diabetes of the Obese.—Attended with the elimination of usually small amounts of sugar.

The *nervous variety:* occurring in cases of neurasthenia, etc.; and finally a *severe variety:* in which the oxidation of glucose is greatly disturbed and albuminous metabolism takes place as a result in increasing degree.

The *etiology* is undetermined, although heredity is of influence.

Symptoms.—There is striking desire for food, in the face of constant loss of strength and weight, with marked thirst, even throughout the night, the mouth being constantly dry; polyuria, from five to eight quarts of pale urine of high specific gravity (above 1020) being excreted daily; the presence of sugar in the urine (see p. 107 *et seq.*); also pruritus, furunculosis, gangrene, impotence, cataract, diabetic neuritis (abolition of knee-jerks, ataxia, sciatica), deposits of thrush in the mouth, diabetic coma (convulsions, somnolence, dyspnea, odor of chloroform from the mouth), acetone-oxybutyric-acid intoxication (iron chlorid reaction, see p. 112). Cases of diabetes manifest a special tendency to pulmonary tuberculosis, nervous disturbances, etc.

Diagnosis.—In all cases of obscure chronic disease the urine should be examined for sugar; and if this be found present, the quantity should be determined, as well as the influence of the food upon its elimination and the amount.

Treatment.—Approximate but not absolute avoidance of all articles of food containing sugar or starch (confections, potatoes, farinaceous foods, beer, sweet wines), with the substitution therefor of food rich in fats and albumin, green vegetables, stewed fruits, little bread, saccharin, together with gymnastic exercises, a course at Carlsbad, baths (Carlsbad, Neuenahr, Vichy), frictions, opium (0.02—grain $\frac{1}{3}$) for thirst.

Diabetes insipidus occurs in nervous individuals and in the sequence of traumatism. It is attended with polyuria and polydipsia. The urine is passed in large amounts, is of low specific gravity and free from sugar. Wasting may be a prominent feature of the disease.

7. *Uric-acid Diathesis* (gout, uratic arthritis).

Deposition of uric acid in the tissues of the body takes place most commonly in the articular cartilages, in the cartilages of the ear, in the tendons and fascia, the skin, and the kidneys.

Among the *etiologic factors* are heredity, alcoholism, obesity, lead-poisoning, gluttony, etc.

In cases of typical *gout* paroxysms are repeated from time to time. These are attended with marked swelling, redness, and painfulness of the great toe (podagra, metatarsophalangeal articulation), the thumb and the joints of the hand (chiragra), the shoulder-joint (omagra, etc.). At the same time there occurs also vomiting, while the appetite is lost. In the further course of the disease deposition of urates takes place (tophi, gouty nodules), giving rise to deformities of joints, together with the development of chronic gastric and intestinal catarrh (visceral gout), bronchial catarrh, and finally to contracted kidney (renal gout).

Treatment.—Restriction of the amount of meat; avoidance of fat and fat-producing food; predominance of vegetables; alcohol is to be interdicted; gymnastics; massage; systematic pedestrian journeys; warm baths (Wiesbaden, Baden-Baden, Teplitz, etc.); moor-baths.

In the gouty attack narcotics may be administered; hot or cold packs employed; cotton batting applied; salophen, etc., administered; later lithium carbonate, urea (10 to 15 grains), lysidin, uricedin, piperazin, etc.

8. *Obesity.*

This condition is frequently hereditary. It occurs in conjunction with indulgence in an excess of food, and particularly of liquids (beer, coffee), with deficient physical activity.

The more troublesome *symptoms* are difficulty in breathing, obstinate constipation, meteorism, intertrigo, excessive diaphoresis. It is often attended with idiopathic hypertrophy of the heart, and frequently with nervous derangement of the action of the heart.

Treatment.—Systematic pedestrianism; restriction of the amount of fluids ingested (coffee, soup, beer, wine, milk); small amounts of farinaceous food, sweets and fat, with a larger amount of meat and vegetables; fruit; a course at Carlsbad, Marienbad, Kissingen, etc.

VII. DISEASES OF THE JOINTS AND BONES.

1. *Rachitis* (English disease).

This occurs between the first and third years of life, and is attended with chronic inflammatory changes in the epiphyseal cartilages, etc., deficient deposition of calcium salts, deformity of bones, usually in conjunction with derangement of gastro-intestinal activity.

Among the *physical signs* are enlargement of the epiphyses; curvature of the extremities; protracted failure of the fontanels to close; delayed dentition; craniotabes (softening of the occipital bones); projection of the sternum and retraction of the ribs (through the action of the diaphragm), giving rise to chicken-breastedness; spinal curvature, pelvic deformity, etc. Usually there exist also anemia, meteorism, etc.

Treatment.—Phosphorus and codliver-oil (0.02 to 150.—gr. ⅓–℥v); sool-baths; nutritious diet; recumbency upon a firm mattress (the deformities often being favored by carrying the children about); abundant exposure in the open air.

2. *Osteomalacia.*

This condition depends upon abnormal absorption of the bone-salts of the pelvis, the vertebral column, the extremities, especially in women (pregnancy, puerperium), and the consequent development of deformities of the skeleton.

The *prognosis* is doubtful.

The *treatment* is the same as that described for rachitis.

3. *Deforming Arthritis* (rheumatoid arthritis).

This occurs in the sequence of repeated attacks of acute articular rheumatism (p. 144), or spontaneously, especially in washerwomen and laborers. It is observed also in cases of tabes dorsalis. It occurs usually at an advanced period of life. The condition is attended with thickening of the articular cartilages, the formation of outgrowths and osteophytes, and villous processes and roughening of the surfaces of the joints. The fingers are deflected outward at the metacarpophalangeal articulations, and, with the hands, are more or less fixed. Pain is present on walking, in consequence of senile changes in the hip-joint.

Treatment.—Massage, sand-baths, moor-baths, electricity, arsenic.

4. *Muscular Rheumatism.*

This occurs principally in the sequence of exposure to cold. For several days at a time severe muscular pain attends all movement. When the muscles of the lower part of the back are affected (*lumbago*) stooping and turning are impossible, and radiating pain is present. *Rheumatic torticollis* (wry-neck) is attended with pain in the muscles at the nape of the neck, interfering with movement. Deep-seated disease is to be excluded (neuritis, tabes, nephritis, bone-disease, etc.), especially when symptoms of chronic rheumatism are present.

Treatment.—Hot packs; steam-bath; mustard-plaster; massage; electricity; salicylates, etc.

VIII. SOME IMPORTANT FORMS OF POISONING.

1. *Acids and Alkalies* (H_2SO_4, HCl, HNO_3, NaOH, KOH, etc.).

Among the results of the action of these are corrosion and destruction of the walls of the mouth, the esophagus, and the pharynx, together with hematemesis, bloody diarrhea, and collapse.

Treatment.—For poisoning with acids: milk, magnesia, chalk, ice. For poisoning with caustic alkalies: vinegar, lemon-juice, mucilaginous mixtures, ether.

Emetics are not to be employed, and the sound should not be introduced into the stomach.

2. *Mercury.*

Poisoning with mercuric chlorid is attended with corrosion, gastro-enteritis, anuria, and collapse.

Treatment.—Milk, albuminous solutions, iron in powder-form; narcotics.

Chronic mercurialism is characterized by ulcerative stomatitis, enteritis, and anemia, together with mercurial tremor.

Treatment.—Withdrawal of mercury, gargles with potassium chlorate, potassium iodid, baths.

3. *Phosphorus.*

This gives rise to a sense of burning in the throat, garlicky eructations and vomiting, with diarrhea and jaundice (fatty degeneration of the liver).

Treatment.—Lavage of the stomach, copper sulphate, oil of turpentine, but no fat.

In cases of chronic phosphorus-poisoning necrosis of the lower jaw often takes place.

4. *Arsenic.*

This gives rise to gastro-enteritis, syncope, cyanosis, collapse.

Treatment.—Lavage, emetics, mixture of magnesia and hydrated iron oxid.

5. *Lead.*

This is attended with the appearance of a blue line at the margin of the gums, together with colicky pain in the abdomen, obstinate constipation, anemia, contracted kidney, wrist-drop from paralysis of the extensors of the hand.

Treatment.—Potassium iodid, sool-baths, electricity.

6. *Alcohol.*

Acute alcoholism may result from an ordinary debauch. Chronic alcoholism is attended with puffiness of the face, bleared eyes, tremulousness of the tongue, tremor of the hands, chronic catarrh of the mucous membranes, hypertrophy of the heart, contracted kidney, cirrhosis of the liver, and neuritis. Delirium tremens is marked by the occurrence of visual hallucinations (of animals) and maniacal states.

Treatment.—Narcotics with care; cool baths.

7. *Chloroform.*

This gives rise to loss of consciousness, abolition of all reflexes, dilatation of the pupils, paralysis of respiration and of the heart.

Treatment.—Artificial respiration, galvanization of the phrenic nerve, douches.

8. *Iodoform.*

This gives rise to vertigo, maniacal attacks, hallucinations, and coma.

Treatment.—Stimulants, baths, atropin.

9. *Carbon Monoxid.*

This gives rise to disturbance of consciousness, roaring in the ears, vomiting, pallor, cyanosis of the face, convulsions, paralysis of respiration, subnormal temperature, albuminuria, and glycosuria. The blood displays the spectrum of CO hemoglobin (see Plate 6).

Treatment.—Artificial respiration, stimulants, transfusion.

10. *Hydrocyanic Acid* (potassium cyanid).

This gives rise to the odor of bitter almonds, retarded respiration, abolition of reflexes, convulsions, paralysis of the heart, and rapid death.

Treatment.—Emetics, artificial respiration, douches, stimulants, hydrated iron oxid, chlorin-water.

11. *Carbolic Acid.*

This gives rise to corrosion, gastritis, coma, paralysis of the heart, nephritis. The urine is dark, and of olive-green color (see pp. 101, 105).

Treatment.—Lavage of the stomach, lime-water, sodium sulphate.

12. *Atropin* (belladonna).

This gives rise to dryness of the throat, thirst, vertigo, hallucinations, mydriasis, tachycardia.

Treatment.—Morphin, physostigmin, pilocarpin.

13. *Digitalis* (foxglove).

This gives rise to vomiting, slowing of the pulse, collapse, and somnolence.

Treatment.—Emetics, lavage of the stomach, tannic acid, stimulants (camphor and ether, black coffee, liquor ammonii anisatus).

14. *Nicotin.*

Acute nicotin-poisoning gives rise to slowing of the pulse, vomiting, salivation, delirium, coma, tetanic attacks. *Chronic nicotin-poisoning* induces palpitation of the heart, asthmatic and anginal attacks, tremor, amblyopia (fluttering scotoma), chronic catarrh, and symptoms resembling those of tabes.

The *treatment* is symptomatic, and the use of tobacco should be forbidden.

15. *Morphin.*

Acute morphin-poisoning is characterized by nausea, vomiting, coma, narrowing of the pupils, slowing of the pulse, and Cheyne-Stokes breathing.

Treatment.—Emetics (zinc sulphate), lavage of the stomach, tannic acid, black coffee, atropin, stimulants, cool baths, artificial respiration.

Chronic morphin-poisoning gives rise to anemia, insomnia, prostration, vertigo, morbid fear, psychoses.

The *treatment* consists in withdrawal of the drug, preferably in an institution, and is in other respects symptomatic.

16. *Ergot.*

Acute ergotism is attended with gastro-enteritis, vertigo, sopor, derangement of respiration.

Treatment.—Emetics, purgatives, tannic acid, stimulants.

Chronic ergotism is attended with symptoms resembling those of tabes; paresthesiæ of the extremities, abolition of the knee-jerks, convulsions, psychoses, gangrene.

The *treatment* is symptomatic.

17. *Toadstool-poisoning.*

This is attended with gastro-enteritis, hemoglobinemia, delirium, epileptiform attacks, narrowing of the pupils, salivation, sopor.

Treatment.—Emetics, drastics, tannic acid, stimulants. The antidote to muscarin is atropin.

18. *Sausage-poisoning and Meat-poisoning* (Botulismus). These are attended with gastro-enteritis, vertigo, pupillary changes, ptosis (in cases of sausage-poisoning), oculomotor palsy, dysphagia, heart-failure, collapse, fever.

Treatment.—Emetics, cathartics, calomel, stimulants, warm baths with douches, nutritive enemata.

When called to a case of poisoning it is best to take with one, in addition to the stomach-tube, the following:

(1) Apomorphin hydrochlorate 0.03 (gr. ss), distilled water 4. (fʒj). Dose: from one-half to a syringeful subcutaneously as an emetic.

(2) Calcined magnesia q.s.; to be taken in teaspoonful doses in water, in cases of poisoning with acids, etc.

(3) Tannic acid 0.5 (gr. vijss); to be divided in ten equal parts, one of which is to be taken every ten minutes, in cases of alkaloidal poisoning.

(4) Four per cent. solution of morphin, of which from one-half to three-quarters of a syringeful may be injected subcutaneously.

(5) Camphor 4. (ʒj), ether 16. (fʒiv); of which from two to four syringefuls may be injected subcutaneously every four hours.

SECTION V.

THERAPEUTIC NOTES.

I. DIETETIC METHODS OF TREATMENT.

1. *Diet for diseases of the stomach*, especially gastric ulcer.

First meal: milk, meat-juice, bouillon, Zwieback, Selters water.

Second meal: eggs soft-boiled or raw, rice, sago boiled in milk, mucilaginous soups, bran, stewed chicken, squab, calves' feet.

Third meal: chipped ham, beefsteak, mashed potatoes, boiled rice, coffee, tea.

Fourth meal: roast beef, roast veal, game (deer, partridge, hare), fish (pike), chicken, broiled squab, wheat-bread, soup, stewed fruit, light farinaceous articles.

Forbidden: fat meat, pork, goose, turkey, sweets, dumplings, potatoes, puddings, liquors, strong spices.

2. *Fever-diet:* milk, eggs, soups, beef-juice, peptones, chicken, squab, calves' feet, jellies, fruit-juices, ice-cream, white wine in Selters water, lemonade.

3. *Rest-cure* (for cases of neurasthenia, anemia, tuberculosis and other chronic diseases):

1. Milk (with possibly the addition of some coffee, tea, or Selters water), from two to four quarts daily at intervals of two hours. As substitutes whey, kefyr, etc., may be employed.

In the intervals small amounts of buttered bread, Zwieback, cold and warm roasts, fowl, game, veal or steak may be given, with green vegetables, farinaceous food, and stewed fruit.

(2) At the beginning complete rest in bed should be

insisted upon. Later, rest in the open air may be permitted. Then, general massage of the whole body should be practised daily for half an hour, and perhaps also faradization of the abdomen.

The *treatment* should be continued from four to six weeks, and is best carried out in an institution.

4. *Reduction-cure* (for obesity).

(*a*) Systematic walking (gradual mountain-climbing), especially in the morning and afternoon, for from two to four hours daily.

(*b*) Restriction of the amount of liquids ingested to between 1500 and 2000 cu. cm.; little soup, coffee, tea, sauces; no beer, wine, or liquor; some cider with Selters water.

(*c*) Little farinaceous food, bread, sugar, confections, fats; more meat of all kinds, green vegetables, lean cheese, fruit.

(*d*) Cold spongings; cold half-baths.

5. *Nutrient Enemata.*

Following a cleansing enema, from 50 to 200 grams of one or other of the following are administered two or three times daily: the yolks of from two to four eggs in mucilaginous soup, milk, 20 per cent. solution of glucose, beef-juice; or Leube's mixture of beef and pancreas: 3 parts of beef, 1 of pancreas chopped, uncooked, into fragments and mixed with two parts of water, possibly with the addition of hydrochloric acid and pepsin.

6. *Artificial Foods* (for emaciated patients).

Condensed milk, cream-mixture, children's foods, meat-extract, meat-solution, beef-juice, beef-peptones, meat-powder.

II. HYDROTHERAPY.

(*a*) *Friction and flagellation* are generally practised morning and evening, the body or only a portion being enveloped in a wet cloth at a temperature of from 15° to 20° (59° to 68° F.) for from two to five minutes. The procedure may be followed by rest or some activity.

(*b*) *Wet packs* in linen cloths also may be applied to the whole body or to only a portion. They are continued from one to five hours and in cases of fever may be renewed hourly. The body is to be subsequently dried.

Sometimes a wet cloth is wrapped about a part and enclosed in an impermeable material, such as gutta percha, taffeta, etc.

(*c*) *Cold douches* are usually directed to individual parts of the body, such as the trunk or the extremities.

(*d*) *Cataplasms* are made with bread, linseed, potatoes, etc., and are employed to alleviate the pain of colic and of inflammation, etc.

(*e*) *Baths* may be full, half, hip and partial.

Steam baths, lasting from fifteen to thirty minutes, with subsequent douche, and *Roman baths*, lasting for an hour, are employed in cases of rheumatism and of dropsy.

Hot baths (above 30° R.—99.5° F.) and *warm baths* (between 26° and 30° R.—90.5°–99.5° F.) are employed in anemic states, in cases of nephritis, during convalescence, etc. They are continued for from twenty to thirty minutes.

Cool baths (from 16° to 22°—68°–81.5° F.) are employed in cases of high fever, of intoxication, and of convulsions, as well as for hardening the system. They are continued for from three to nineteen minutes.

Medicated Baths.

Addition of *salt* (sodium chlorid, bathing salt, from 5 to 10 lbs.), in cases of anemia, scrofulosis, rachitis, etc.

Moor-baths (about 50 lbs. of peat-soil or bog-earth, or from 5 to 10 lbs. of moor extract), in cases of rheumatism, gout, neuralgia, etc.

Sool-baths (from 2 to 5 quarts of brine, sool salts), in scrofulosis, anemia, gout, etc.

Pine-needle baths (from 100. to 200. of pine-needle extract); *bran-baths* (5 lbs. of wheat bran); *sand-baths* (warm, dry sea-sand); in cases of chronic arthritis, etc.

III. TREATMENT BY CLIMATE AND BATHS.

Among numerous health-resorts the following may be mentioned:

(1) For diseases of the *respiratory organs:* Ems, Selters, Reichenhall, Giesshübl, Soden, Vichy, Neunahr, Assmannshausen, Wiesbaden, Kissingen, Sylt, Norderney, etc.

Especially for *pulmonary tuberculosis:* Falkenstein, Görbersdorf, St. Blasien, Meran, St. Moritz, Davos, Montreaux, Mentone, Riviera, etc.

(2) For *diseases of the heart:* Nauheim, Oeynhausen, Homburg, Wiesbaden, Baden-Baden, elevated resorts, sea-baths.

(3) For *disorders of the stomach and intestines:* Kissingen, Neuenahr, Bilin, Wiesbaden, Baden-Baden, Homburg, Carlsbad.

(4) For *diseases of the bodily metabolism* (obesity, gout, etc.): Marienbad, Carlsbad, Franzensbad, Friedrichshall, Elster, Ofen, Tarasp, etc.

(5) For cases of *anemia*, etc.: Schwalbach, Brückenau, Steben, Alexandersbad, Franzensbad, Elster, Pyrmont, etc.

(6) For *diseases of the kidneys and bladder:* Fachingen, Wildungen, Vichy.

(7) For *diseases of the liver* (gall-stones): Carlsbad, Kissingen, Franzensbad, Ofen, etc.

(8) For *diseases of the joints* (neuralgia, etc.): Brückenau, Aibling, Homburg, Franzensbad, Assmannshausen, Baden-Baden, Wiesbaden, Kissingen, etc.

IV. PHYSICAL METHODS OF TREATMENT.

1. *Electricity.*

This is frequently employed in the form of the galvanic or the faradic current.

The former is believed to exert a more profound influence than the latter. Painful (the anode is sedative, the kathode stimulating) and central disorders yield to galvanism, peripheral disorders, and especially motor palsies to faradism. Sometimes frictional or static electricity is employed.

Too strong currents should not be employed, and a reliable galvanometer should be used.

2. *Massage.*

This may take the form of stroking, kneading, hacking and rolling, daily, for from a quarter to half an hour, preceded by inunction with oil or petrolatum, and followed by warm coverings. It is useful in the treatment of painful diseases of the muscles, bones and joints, chronic rheumatism, nervous diseases, anemia, etc.

3. *Gymnastics.*

This includes the intelligent pursuit of such exercises as bicycling, rowing, turning, pedestrian tours, ball-playing, and the like; as well as home-gymnastics with various forms of apparatus, for half an hour daily before eating; and finally resistance-gymnastics with apparatus, for an hour or two daily.

Pneumotherapy.—Respiration in rarefied or compressed air, possibly in conjunction with inhalation of various medicaments (sodium chlorid, sool, ethereal oils, etc.), is employed in various sanatoria, in the treatment of asthma, emphysema, pulmonary tuberculosis, chronic bronchitis, etc.

V. THE MOST IMPORTANT MEDICAMENTS.

1. *Antipyretics*, *Antirheumatics*, etc.:

Quinin hydrochlorate,	0.5–2.	(gr. vijss–gr. xxx)	in	cachets,	twice or thrice daily.
Sodium salicylate,	2.–5.	(gr. xxx–gr. lxxv)	"	"	twice daily.
Salicylic acid,	0.5	(gr. vijss)	"	"	6 to 10 times daily.
Salol,	2.	(gr. xxx)	"	"	3 or 4 times daily.
Salophen,	1.–2.	(gr. xv–gr. xxx)	"	"	twice or thrice daily.
Salipyrin,	1.–2.	(gr. xv–gr. xxx)	"	"	2 to 4 times daily.
Antipyrin,	1.	(gr. xv)	"	"	2 to 5 times daily.
Antifebrin,	0.20–0.5	(gr. iij–gr. vijss)	"	"	twice or thrice daily.
Phenacetin,	0.5–1.	(gr. vijss–gr. xv)	"	"	3 or 4 times daily.

2. *Acids:*

Dilute hydrochloric acid, from 5 to 10 drops in water, thrice daily; from 1 to 2 per cent. of lemon-juice in various mixtures.

3. *Expectorants:*

Infusion of ipecac-root 0.3–0.5 (gr. v–gr. vijss) to 120. (f℥iv); tablespoonful every two hours.

Infusion of senega-root, 10. (ʒijss) to 120. (f℥iv); tablespoonful every two hours.

Liquor ammonii anisatus, 3 per cent., or in drops from five to ten thrice daily.

Ammonium chlorid, 5. (gr. lxxv) to 120. (f℥iv); from a dessertspoonful to a tablespoonful every two hours.

Apomorphin, 0.03–0.05 (gr. ss–gr. ¾) to 150. (f℥v); tablespoonful every two hours.

Benzoic acid, 0.1–0.3 (gr. jss–gr. ivss), in cachets, every three hours.

Potassium iodid, 5. (gr. lxxv) to 150. (f℥v); tablespoonful thrice daily.

Balsam of Peru, oil of turpentine, myrtol, creosote, etc., in gelatin capsules of from 0.1 to 0.5 (gr. jss–gr. vjss), several times daily.

4. *Inhalants:*

Sodium chlorid, 1 per cent.

Sodium bicarbonate, 1 per cent.

Ammonium chlorid, 1 per cent.

Ems water, etc.

Potassium bromid, 2 per cent.

Tannic acid, 2 per cent.

Lime-water, 50 per cent.

Turpentine, balsam of Peru, cherry-laurel water, etc., in drop-doses.

5. *Topical Applications:*

Iodin, 0.5 (gr. vijss); potassium iodid, 2.5 (gr. xxxvijss); glycerin, 25. (fʒvj).

Borax and glycerin, 5. (gr. lxxv) to 25. (fʒvj).

Silver nitrate, from 1. to 10. (gr. xv–ʒijss) to 50. (f℥jss).

6. *Gargles:*

Potassium chlorate, 2 per cent.

Alum, from 3 to 5 per cent.

Borax, 3 per cent.

Potassium permanganate, from 0.1 to 0.5 per cent.

Hydrogen dioxid, 2 per cent.

7. *Cardiants* (see pp. 181, 182).

Powdered digitalis-leaves, 0.1 to 0.15 (gr. jss–gr. ijss), in cachets, every two hours; or infusion, 1. or 2. (gr. xv–xxx) to 120. (f℥iv).

Caffein, 0.2 (gr. iij), in cachets, three or four times daily.

Tincture of strophanthus, from 10 to 15 drops thrice daily.

Camphor and ether 5. (gr. lxxv) to 20. (f ʒv); two or three syringefuls subcutaneously at hourly intervals.

8. *Stomachics:*

Dilute hydrochloric acid, from five to ten drops in water several times daily.

Bitter tincture, compound tincture of cinchona, a teaspoonful thrice daily.

Tincture of rhubarb, a teaspoonful thrice daily.

Sodium bicarbonate, 30. (℥j); bismuth subnitrate, 1.5 (gr. xxijss); from ten to twenty grains every two hours.

9. *Emetics:*

Apomorphin, 0.05 (gr. ¾) to 5. (♏lxxv); from a half to one syringeful subcutaneously.

Copper sulphate, 1. (gr. xv) to 50. (f℥jss); one or two teaspoonfuls.

10. *Laxatives:*

Castor-oil, one or two teaspoonfuls; may also be given in capsules.

Carlsbad salt (sodium sulphate, 50. (℥jss); sodium bicarbonate, 6. (ʒjss); sodium chlorid, 3. (gr. xlv); dose: a teaspoonful.

Magnesium sulphate, 50. (℥jss); sodium bicarbonate, 10. (ʒijss); dose: from ten to twenty grains.

Magnesium sulphate, 30. (℥j); powdered rhubarb-root, 10. (ʒijss); dose: from ten to twenty grains.

Calomel. 0.3 to 0.5 (gr. v–gr. vijss), three or four times daily.

Infusion of senna-leaves, 10. (ʒijss) to 120. (f℥iv).

Extract of aloes, extract of rhubarb, of each 5. (gr. lxxv), with powder and juice of licorice sufficient to make 100 pills; one twice or thrice daily.

Glycerin enemata, 5. to 20. (♏lxxv–f℥v); oil enemata, etc.; suppositories; enemata of simple water.

11. *Astringents:*

Infusion of calumba-root, 15. (ʒiv) to 150. (f℥v); tablespoonful at intervals of two hours.

Decoction of salep-root, 1. (gr. xv) to 150. (f℥v); tablespoonful at intervals of two hours.

Gum-mixture, at hourly intervals.

Tannic acid, 0.05 to 0.1 (gr. ¾–gr. ijss) to 100. (℥iijss).

Bismuth subnitrate or salicylate, from 0.5 to 2. (gr. vijss–gr. xxx) to 100. (f℥iijss).

Opium, extract of opium, from 0.02 to 0.04 (gr. ⅓–gr. ss), from three to five times daily.

Tincture of opium, from 10 to 20 drops, from three to five times daily.

Tincture of opium, from 10 to 20 drops, from three to five times daily.

12. *Anthelmintics:*

Santonin, from 0.03 to 0.05 (gr. ½–gr. ¾), two or three times daily.

Calomel, from 0.05 or 0.1 to 0.3 (gr. ¾–gr. jss–gr. v), three or four times daily.

Extract of filix mas recently prepared, from 10. to 12. (ʒijss–ʒiij), in capsules each containing 2. (gr. xxx).

13. *Diuretics* (see Digitalis):

Calomel, 0.2 (gr. iij), three or four times daily.

Solution of potassium acetate, 20. (ʒv) to 120. (f℥iv).

Squill, from 0.05 to 0.3 (gr. ¾–gr. v), from three to five times daily.

Potassium and sodium borotartrate, from 10. to 15. (ʒijss–ʒiv) to 120. (f℥iv).

Theobromin sodiosalicylate, 2. (gr. xxx), from three to five times daily.

Special diuretics (juniper-berries, petroselinum, ononis-root,

14. *Diaphoretics:*

Pilocarpin hydrochlorate, 0.2 (gr. iij) to 10. (fʒijss); dose: one-half to one syringeful subcutaneously.

Special diaphoretics (linden-flowers, elder-flowers, chamomile-flowers).

15. *Narcotics, hypnotics:*

Morphin hydrochlorate, from 0.01 to 0.03 (gr. $\frac{1}{6}$–gr. ss), from three to five times daily; or 0.1 (gr. jss) to 10. (fʒijss); dose: one syringeful subcutaneously.

Chloral hydrate, from 2. to 3. (gr. xxx–gr. xlv), in cachet.

Chloralamid, 2. (ʒss) in cachet.

Sulfonal, from 1.5 to 2. (gr. xx–gr. xxx), in cachet.

Trional, from 1.5 to 2. (gr. xx to gr. xxx) in cachet, followed by a cup of tea, etc.

Potassium bromid, from 2. to 5. (gr. xxx–gr. lxxv), in milk.

Cocain hydrochlorate, from 0.5 to 1.5 (gr. vijss–gr. xxijss) to 10. (fʒijss), for topical application.

16. *Roborants*, etc.:

Blaud's pill (iron sulphate, potassium carbonate, each 15. (ʒiv); tragacanth, sufficient to make 100 pills). Dose: from one to three pills thrice daily.

Sirup of iron iodid, from a quarter to a half teaspoonful thrice daily; best given in extract of malt.

Saccharated iron oxid, from gr. v.–gr. x, every four hours.

Solution of iron albuminate, a teaspoonful thrice daily.

Ferratin, 0.1 (gr. jss), thrice daily.

Quinin sulphate, from 0.03 to 0.1 (gr. ss–gr. jss), in pill; dose: from three to five daily.

Arsenous acid, from 0.0025 to 0.004 (gr. $\frac{1}{30}$–gr. $\frac{1}{16}$), in pills; dose: from three to five daily.

Solution of potassium arsenite, from three or five to eight drops thrice daily.

Extract of ergot, 0.5 (gr. vijss) in pills; dose: from three to five daily.

Atropin sulphate, 0.0005 (gr. $\frac{1}{140}$) in pill, thrice daily.

In writing prescriptions complex formulæ should be avoided, as it is difficult to analyze the results of their activity, and the cost is unnecessarily increased.

INDEX.

ABDOMEN, 5, 14
  distention of, 94
  examination of, 93
  exploratory puncture of, 15
  form of, 14
  measurement of, 15
  palpation of, 15
  percussion of, 16
  retraction of, 94
  symmetry of, 15
Abscess of liver, 208
  old, hematoidin-crystals from, Pl. 21
  pulmonary, 171
  retropharyngeal, 81, 193
Abscesses, contents of, Pl. 21
Accentuation of heart-sounds, 64
Accidental murmurs, 66
Acetone in urine, 112
Achorion schœnleinii, 121
Acidity, total, of gastric juice, 83
    determination of, 86
Acidophile cells, 73
Acids, 237
  poisoning with, 228
    treatment of, 228
Actinomyces-fungus, 125
  -granules in sputum, 54
  -infections, 150
Actinomycosis, 150
  treatment of, 150
Adenoid vegetations of the naso-pharynx, 193
    treatment of, 193
Adult life, diseases of, 2
Advanced life, diseases of, 2
Age, 1
Albumin in urine, 105
  tests for, 106
Albuminous bodies, 115
Albuminuria, febrile, sediment from, Pl. 19
  physiologic, 105
Albumoses, 107
Alcohol, poisoning with, 229
    treatment of, 230
Alkalies, poisoning with, 228
    treatment of, 228
Ammonia in urine, 103
Ammonio-magnesium phosphate in urine, Pl. 1
Ammonium urate in urine, Pl. 14
Amœba coli, 130
Amphophile cells, 74
Amphoric breathing, 47
Amyloid degeneration of liver, 209
  kidney, 215
    sediment from, Pl. 19
Anadicrotism, 70
Anemia, 220
  acute, 220
    symptoms of, 220
    treatment of, 220
  chronic secondary, 221
    blood in, 221
    prognosis of, 221
    symptoms of, 221
    treatment of, 221
  climatic treatment of, 236
  pernicious, 221
    etiology of, 221
    treatment of, 221
  simple profound, Pl. 3
Aneurism of the aorta, Pl. 50, p. 190
Angina, catarrhal, 192
  diphtheric, 192
  lacunar, 192
  Ludwig's 192
  necrotic, 192
  parenchymatous, 193
  pectoris, 186
    treatment of, 186
  simple, 192
    treatment of, 193
Angle, epigastric, 13
Anguillula intestinalis, 127

Ankylostomum, 202
  duodenale, 126
Anthelmintics, 240
Anthrax, 143
  treatment of, 143
  -bacilli, 26, 123, 143
    in sputum, 54
Antipyretics, 237
Antipyrin in urine, Pl. 21
Antirheumatics, 237
Aorta, aneurism of, Pl. 50, p. 190
    prognosis of, 190
    treatment of, 190
Aortic insufficiency, Pl. 46
  orifice, stenosis of, 59
  valve, insufficiency of, 58
Apex-beat, 61
  augmentation of, 62
  displacement of, 62
  enfeeblement of, 62
Aphthæ, 191
  Bednar's, 191
Appetite, 5
Argas reflexus, 126
Arsenic, poisoning with, 229
    treatment of, 229
Arteriosclerosis, 189
  prognosis of, 190
  sequelæ of, 189
  treatment of, 190
Arthritis, deforming, 227
    treatment of, 228
Artificial foods, 234
Arytenoid muscle, transverse, paralysis of, 39
Ascarides, 202
Ascaris lumbricoides, 126
Ascites, 91, 94, 204
  attending cardiac lesion, Pl. 62
  due to stasis, 95
  inflammatory, 95
  symptoms of, 204
  treatment of, 205
Aspergillus fumigatus, 121
  -mycelium in sputum, 54
Asthma, bronchial, 167
    sputum from, Pl. 8
    treatment of, 167
Astringents, 240
Atelectasis, 168
  treatment of, 168
Atropin, poisoning with, 230
    treatment of, 230
Auscultation, 31
  of cardiac orifices, 14
Auscultation of heart, 64
  of lungs, 14, 46
  of voice, 48
Auscultatory topography, Pl. 24

BACILLI, 123
Back, percutory topography of, Pl. 26
Bacteria, 132
Bacterial infection, 131
Bacteriologic methods of investigation, 24
Bacterium coli commune, Pl. 22, p. 125
  lactis ærogenes, 125
Basophile cells, 73
  granulation, Pl. 2
Baths, 235
  bran, 235
  cool, 235
  hot, 235
  medicated, 235
  moor, 235
  pine-needle, 235
  Roman, 235
  sand, 235
  sool, 235
  steam, 235
  treatment by, 236
  warm, 235
Bednar's aphthæ, 191
Biermer's change in percussion-note, 45
Biliary acids in urine, 112
  coloring-matter, Pl. 20
    in urine, 112
Biot's breathing, 13
Bismuth-test, Pl. 20
Bladder, 100
  acute catarrh of, 218
      symptoms of, 218
      urine in, 218
  chronic catarrh of, 219
  diseases of, 218
    climatic treatment of, 236
    urinary sediments attending, Pl. 18
  squamous epithelium from, Pl. 16
  tumors of, 219
    diagnosis of, 219
    symptoms of, 219
    treatment of, 219
Bleeders' disease, 224
Blood, 16, 72
  arterial, 72

Blood, diseases of, 220
  in stools, 97
  in urine, 111
  in various diseases, Pl. 3
  microscopic examination of, 77
  normal, fresh preparation of, Pl. 1
    frog's, stained, Pl. 1
    stained preparation of, Pl. 1
  parasites of, Pl. 5
  spectroscopy of, 74
  venous, 72
  -casts, Pl. 17
  -corpuscles, enumeration of, 75
    red, Pl. 1, 16, p. 72
      abnormal variations in form of, Pl. 1
        in size of, Pl. 1
      nucleated, of man, Pl. 1
    white, Pl. 2, p. 73
  -crystals, Pl. 6
  -plates, 74
  -preparation from a case of typhoid fever, Pl. 3
  -spectra, Pl. 6
  -state of leukemia, Pl. 14
  -test, Heller's, Pl. 20
  -vessels, examination of, 66
    systolic murmurs over, 66
Bloody sediment, Pl. 13
Bodily development, 7
  temperature, 9
  vigor, 4, 7
Body, projection of internal organs upon anterior aspect of, Pl. 23
    upon posterior aspect of, Pl. 25
Bones, diseases of, 227
Bothriocephalus latus, 129
Botulismus, 232
Bowel, carcinoma of, 201
  occlusion of, Pl. 64, p. 201
  stenosis of, 201
Bowels, 6
  evacuation of, pain in, 18
Breakfast, trial, 87
Breathing, amphoric, 47
  Biot's 13
  bronchial, 46
  Cheyne-Stokes, 13
  metamorphosing, 47
  undefined, 47
  vesicular, 46
Breath-sounds, absence of, 47
Brickdust sediment in urine, Pl. 13
Bromin in urine, Pl. 21
Bronchial breathing, 46
  respiratory murmur, 32
Bronchiectasis, 166
  treatment of, 167
Bronchitis, acute, 165
    treatment of, 165
  acute diffuse, Pl. 35
  chronic, 165
    treatment of, 165
  croupous, 166
    treatment of, 166
  fetid, 166
    treatment of, 166
Bronchoblennorrhea, 165
Bronchopneumonia, Pl. 36, p. 168
  treatment of, 168
Buccal mucous membrane, 12
Budding fungi, 121

Calcium carbonate, Pl. 14
    in urine, 103
  oxalate, Pl. 14
    calculi of, 114
  phosphate, Pl. 14
  sulphate, Pl. 14
Calculi, calcium-oxalate, 114
  cystin, 114
  in stools, 98
  of carbonates, 114
  phosphatic, 114
  renal and vesical, examination of, 114
  uric acid, 114
  xanthin, 114
Calculous pyelitis, sediment from, Pl. 18
Carbohydrates, 117
Carbolic acid, poisoning with, 230
    treatment of, 230
Carbon monoxid, poisoning with, 230
    treatment of, 230
Carbonates, calculi of, 114
  in urine, 103
Carcinoma of the bowel, 201
    treatment of, 201
  of the cardia, with stenosis of the esophagus, Pl. 51
  of the pylorus, dilatation of the stomach with, Pl. 53
  of the stomach, Pl. 54, p. 197
Cardia, carcinoma of, with stenosis of the esophagus, Pl. 51

- Cardiac lesion, ascites attending, Pl. 62
  - cells in sputum, 52
  - uncompensated, Pl. 43
- Cardiants, 239
- Catadicrotism, 70
- Cataplasms, 235
- Catarrh, dry, 165
- Cellular elements in sputum, 52
- Cestodes, 128
- Character of pain, 16
- Charcot-Leyden crystals in sputum, 53
- Chest, 5
  - new-growths in, 176
- Cheyne-Stokes breathing, 13
- Chicken-pox, 157
- Childhood, diseases of, 1
- Chill, 5
- Chlorids in urine, 102
- Chloroform, poisoning with, 230
  - treatment of, 230
- Chlorosis, 220
  - blood in, 220
  - complications of, 220
  - diagnosis of, 220
  - treatment of, 220
- Cholelithiasis, Pl. 58, p. 206
  - diagnosis of, 206
  - prognosis of, 206
  - symptoms of, 206
  - treatment of, 206
- Cholera asiatica, 142
  - complications of, 142
  - diagnosis of, 142
  - incubation of, 142
  - intestinal contents of, Pl. 11
  - treatment of, 142
  - morbus, 158
    - diagnosis of, 158
    - treatment of, 158
  - nostras (see Cholera Morbus), 158
    - intestinal contents of, Pl. 11
  - -bacilli, 27, 124, 142
- Cholesterin in urine, Pl. 15
  - -plates in sputum, 53
- Cicatrices, 8
- Circulatory apparatus, examination of, 54
  - organs, diseases of, 177
- Cirrhosis of the liver, Pl. 56, p. 207
- Clearness on percussion, 30
- Climate, 3
  - treatment by, 236
- Coagulum, fibrinous, Pl. 9
- Coccus-infections, 144
- Colicky pain, 17
- Colon, Pl. 23, 27
- Color, 7
  - -reactions of the gastric juice, Pl. 12
    - of the urine, Pl. 20
- Compensation, derangement of, 60
- Conditions, special, 11
- Connective tissue, subcutaneous, 7
- Consciousness, 6
- Constipation, 98
  - habitual, 202
    - treatment of, 202
- Constitution, 7
- Constricted liver, 209
- Continued fever, 9
- Coryza, 161
- Cough, 50
  - laryngeal, 37
- Creatinin in urine, 105
- Crepitant râles, 47
- Crico-arytenoid, posterior, paralysis of, 39
- Croup, 141
- Croupous bronchitis, 166
  - pneumonia, Pl. 29, 30, p. 147, 167
- Cryptogenetic septicopyemia, 146
- Crystals in sputum, 53
- Cultivation, 26
- Curschmann's spirals in sputum, 53
- Cutaneous emphysema, 9
- Cylindroids, Pl. 17
- Cystin, Pl. 15
  - calculi, 114
- Cystitis, 218
  - diagnosis of, 219
  - sediment from, Pl. 18
  - treatment of, 219
  - tuberculous, 219
    - pus from, Pl. 19
    - treatment of, 219
- Cysts, contents of, Pl. 21

- DEGENERATION, amyloid, Pl. 59
- Deglutition, difficulty in, 17
- Demodex folliculorum, 126
- Development, bodily, 7
- Diabetes, ferric-chlorid reaction in, Pl. 20
  - insipidus, 225
  - mellitus, 224
    - diagnosis of, 225
    - etiology of, 225

Diabetes mellitus, symptoms of, 225
    treatment of, 225
  nervous, 224
  of the obese, 224
  severe, 224
Diacetic acid in urine, 112
Diaphoretics, 241
Diaphragm, Pl. 23, 27
Diarrhea, 98
Diazo-reaction, Pl. 20, p. 113
Dicrotism, 70
Diet for diseases of stomach, 233
Dietetic methods of treatment, 233
Digestion, gastric, 85
Digestive apparatus, 79
  organs, diseases of, 190
Digitalis, poisoning with, 230
    treatment of, 231
Dilatation of the stomach, with carcinoma of the pylorus, Pl. 53
Diphtheria, 140
  diagnosis of, 141
  sequelæ of, 141
  treatment of, 141
  -bacilli, Pl. 7, p. 27, 124, 140
  -membrane, Pl. 7
Diphtheric angina, 192
Diplococcus of Fraenkel, 122
Displacement of lung, symptoms of, 44
Distoma hæmatobium, 129
  hepaticum, 129
  pulmonale, 129
Dittrich's plugs in sputum, 53
Diuretics, 240
Diverticula, esophageal, 82
Division of the first sound of the heart, 65
Double sound over femoral artery, 67
Douches, cold, 235
Dulness, cardiac, abnormal, 63
    absolute, 63
    relative, 63
  on percussion, 30
    of lungs, 43
Duodenal ulcer, 197
Duplication of the first sound of the heart, 65
Duroziez's double sound, 67
Dwelling-houses, situation of, 3
Dysentery, 158
  complications of, 158
  prognosis of, 158
  treatment of, 158
Dyspepsia, nervous, 199
    diagnosis of, 199
    treatment of, 199
Dyspnea, 13
  laryngeal, 37

ECHINOCOCCUS of liver, 208
  -fluid, Pl. 21
  -hooklets in sputum, 53
Edema, 8
  of larynx, 164
  pulmonary, 170
    symptoms of, 170
    treatment of, 170
Elastic fibers in sputum, 53
Electricity, 236
Emaciation, 4
Embolism, pulmonary, 171
    symptoms of, 171
    treatment of, 172
Emetics, 239
Emphysema, cutaneous, 9
  pulmonary, Pl. 38, p. 169
    complications of, 169
    treatment of, 170
Endocarditis, acute, 145, 177
    symptoms of, 177
    treatment of, 180
  benign, 177
  malignant, 177
  recurring, 177
  ulcerative, 177
  verrucose, 177
Enemata, nutrient, 234
English disease, 227
Enteritis, acute, 200
    treatment of, 200
  membranous, 200
Enuresis, nocturnal, 219
    treatment of, 220
Eosinophile cells, Pl. 2, p. 73
    in sputum, 52
Epigastric angle, 13
Epiglottis, paralysis of, 40
Epistaxis, 35, 162
Epithelial cells in pelvis of kidney, Pl. 16
Epithelium, renal, Pl. 16
  squamous, from urethra and bladder, Pl. 16
Ergot, poisoning with, 231
    treatment of, 231
Erysipelas, 150
  prognosis of, 150
  treatment of, 150

Erythrocytes, Pl. 1, p. 72, 77
Esbach's test for albumin, 106
Esophageal diverticula, 82
Esophagus, Pl. 25, p. 12, 81
  carcinoma of the cardia, with stenosis of the, Pl. 51
  diseases of, 194
  narrowing of, 81
  stenosis of, 194
    treatment of, 195
Examination of patient, 1
  with mirrors, 22
Exanthemata, 8
  acute, 153
Expectorants, 238
Expectoration, 50
Expiration, prolongation and accentuation of, 46
Exploratory puncture, 23
Extrapericardial murmurs, 66
Extremities, 6, 15
  pain in, 18
Exudates, pleural, 49
Eyes, 11

FACE, pain in, 17
Fat in urine, 113
Fats, 117
Fatty acids in sputum, 53
  casts, Pl. 17
Febrile albuminuria, sediment from, Pl. 19
  symptoms, 5, 9
Feces, 16
  general view of, Pl. 11
Fehling's solution, titration with, for sugar, 110
Femoral artery, double sound over, 67
Fermentation-test for sugar, 108, 110
Ferric-chlorid reaction in diabetes, Pl. 20
Fever, continued, 9
  high, 9
  intermittent, 9
  moderate, 9
  remittent, 9
  slight, 9
  -diet, 233
Fibrinous coagula in sputum, 51
Filaria medinensis, 127
  sanguinis, 127
Flagellation, 234
Food, constituents of, 118
Foods, artificial, 234
Form of abdomen, 14
Fremitus, pectoral or vocal, 48
Friction, 234
  pericarditic, 34
  peritonitic, 34
  pleuritic, 32
  -sounds, pleuritic, 48
Friedreich's respiratory change in percussion-note, 45

GALL-BLADDER, hydrops of, Pl. 58
Galloping rhythm, 65
Gangrene, pulmonary, 170
    complications of, 170
    symptoms of, 170
    treatment of, 170
Gargles, 238
Gastric catarrh, acute, 195
    symptoms of, 195
    treatment of, 195
  contents, 16
    general view of, Pl. 11
  digestion, 85
  juice, 83
    color-reactions of, Pl. 12
  ulcer, 196
    diagnosis of, 196
    symptoms of, 196
    treatment of, 197
Gastritis, acute, 195
  chronic, 195
    diagnosis of, 195
    treatment of, 195
  phlegmonous, 196
General manifestations, 6
Gerhardt's postural change in percussion-note, 45
Gingival deposit, Pl. 7
Glanders, 143
  bacillus of, Pl. 22, p. 143
    treatment of, 144
  -bacilli, 124
Glandular cicatrices, 12
  swellings, 12
  tuberculosis, 134
Glossitis, 192
  dissecting, 192
  parenchymatous, 192
  treatment of, 192
Glottis, spasm of, 164
    treatment of, 164
Glucose, 107
  tests for, 107
Glycosuria, 107, 224

Glycosuria, alimentary, 224
Gmelin's test for bile, 112
Gonococcus, 123
Gonorrhea, pus from, Pl. 19
Gout, 226
  treatment of, 226
Gouty nodule, uric acid from, Pl. 21
Granular tube-casts, Pl. 17
Gregarines, 130
Gums, 12, 79
Gymnastics, 237

HABITUS, 7
Head, 5, 11
Headache, 17
Health, previous, 3
Heart, Pl. 23, 27, p. 14
  anatomy of, 54
  auscultation of, 64
  congenital lesions of, 183
      prognosis of, 184
  diseases of, 177
    climatic treatment of, 236
  examination of, 54
  fatty, 186
    symptoms of, 186
    treatment of, 186
  idiopathic hypertrophy of, Pl. 48, p. 184
      symptoms of, 185
      treatment of, 185
  inspection of, 14
  nervous palpitation of, 186
      treatment of, 186
  neurotic disorders of, 186
  palpation of, 14
  percussion of, 14, 62
  physiology of, 54
  valvular lesions of, 180
      diagnosis of, 181
      theory of, 55
      treatment of, 181
  -murmurs, 33, 65
    diastolic, 66
    systolic, 65
  -sounds, accentuation of, 64
    metallic, 65
Height, 7
Heller's blood-test, Pl. 20
  test for blood, 112
Hematoidin-crystals, Pl. 6
  from an old abscess, Pl. 21
  in sputum, 53
Hematuria, 111
Hemin-crystals, Pl. 6
Hemoglobin, estimation of, 75
Hemoglobinemia, Pl. 3, p. 223
  prognosis of, 223
  symptoms of, 223
  urine of, 223
Hemoglobinuria, 111
Hemopericardium, 188
  symptoms of, 188
Hemophilia, 224
Hemorrhages, 8
Hemorrhagic diathesis, 223
    etiology of, 223
Hemorrhoids, 202
Hemothorax, 176
Heredity, 2
High-pitched tympanitic note, 30
Hippuric acid, Pl. 14
    in urine, 105
History, 1
Hodgkin's disease, 223
    blood of, 223
    treatment of, 223
Hum, venous, 34
Hyaline tube-casts, Pl. 17
Hydrochloric acid, 3
    determination of, 85
Hydrocyanic acid, poisoning with, 230
      treatment of, 230
Hydrogen sulphid in urine, 113
Hydronephrosis, 217
Hydropericardium, 188
  symptoms of, 188
  treatment of, 188
Hydrophobia, 160
  hydrophobic stage of, 160
  incubation of, 160
  prodromes of, 160
  treatment of, 160
Hydrops of the gall-bladder, Pl. 58
Hydrotherapy, 234
Hydrothorax, Pl. 34, p. 176
  symptoms of, 176
  treatment of, 176
Hyperpyretic temperature, 9
Hypertrophy of the heart, idiopathic, Pl. 48, p. 184
Hypnotics, 241
Hypoxanthin in urine, 105

INDICAN in urine, 104
Indican-test, Pl. 20
Infancy, diseases of, 1

- Infarction, hemorrhagic, of kidney, 215
    - of lung, 171
  - pulmonary, Pl. 37
- Infectious diseases, 131
    - of undetermined origin, 153
- Influenza, 141
  - complications of, 142
  - treatment of, 142
  - -bacilli, 124, 141
    - in sputum, 54
  - -bronchitis, sputum from, Pl. 10
- Inhalants, 238
- Inoculation, 28
- Insects, 126
- Inspection of heart, 14
  - of lungs, 42
- Instruments for microscopy, 20
- Insufficiency, aortic, Pl. 46
  - of aortic valve, 58
  - of mitral valve, 56
  - of tricuspid valve, 59
  - tricuspid, Pl. 47
- Intelligence, 6
- Intermittent fever, 9
- Internal organs, projection of, upon anterior aspect of the body, Pl. 23
      - upon lateral aspects of trunk, Pl. 27
      - upon posterior aspect of body, Pl. 25
    - special diagnosis of diseases of, 35
- Intestinal catarrh, 199
    - acute, 190
      - symptoms of, 199
      - treatment of, 200
    - chronic, 200
      - symptoms of, 200
      - treatment of, 200
  - contents of cholera Asiatica, Pl. 11
      - nostras, Pl. 11
  - murmurs, 34
  - parasites, 202
  - tuberculosis, 134, 200
- Intestines, diseases of, 199
    - climatic treatment of, 236
  - microscopy of, Pl. 11
- Invagination of the bowel, Pl. 64
- Investigation, bacteriologic methods of, 24
  - methods of, 19
- Iodin in urine, Pl. 21
- Iodoform, poisoning with, 230
    - treatment of, 230
- Ixodes, 126

- JAUNDICE, 90
  - catarrhal, 205
    - prognosis of, 205
    - treatment of, 205
  - chronic, 206
- Jerking inspiration, 46
- Joints, diseases of, 227
    - climatic treatment of, 236
- Jugular vein, pulsation of, 12

- KIDNEY, 99
  - amyloid, 215
    - etiology of, 215
    - sediment from, Pl. 19
    - urine of, 215
  - cyanotic 214
    - treatment of, 215
  - hemorrhagic infarction of, 215
  - pelvis of, epithelial cells from, Pl. 16
  - sarcoma of the, Pl. 68
  - table of diseases of, 215
  - true contracted, 213
      - diagnosis of, 214
      - sediment from. Pl. 19
      - treatment of, 214
      - urine of, 214
  - tuberculosis of, 217
    - symptoms of, 217
    - treatment of, 217
  - tumors of, 217
  - wandering, 218
    - diagnosis of, 218
    - treatment of, 218
- Kidneys, Pl. 25
  - diseases of, 211
    - climatic treatment of, 236
    - urinary sediments attending, Pl. 18
  - percussion of, 99

- LARYNGEAL dyspnea, 37
  - stenosis, 141
  - tuberculosis, 134
- Laryngismus stridulus, 164
- Laryngitis, acute, 163
    - treatment of, 163
  - chronic, 163
    - treatment of, 163
- Larynx, 12
  - edema of, 164

Larynx, edema of, treatment of, 164
  examination of, 36
  syphilis of, 164
    treatment of, 164
  tumors of, 164
    treatment of, 165
Lavage, 87
Laxatives, 239
Lead, poisoning with, 229
    treatment of, 229
Leprosy, 143
  mutilating, 143
  tuberous, 143
  -bacilli, 124, 143
Leptothrix buccalis, 121
Leucin, Pl. 15
  -spheres in sputum, 53
Leukemia, 221
  acute, Pl. 4, p. 223
  blood of, 222
  blood-state of, Pl. 4
  lymphatic, Pl. 4
  splenomedullary, Pl. 2, 60, p. 222
    blood from, Pl. 4
  treatment of, 223
Leukocytes, Pl. 2, p. 73, 78
  in sputum, 52
  mononuclear, Pl. 2
  polynuclear, Pl. 2
Leukocytosis, Pl. 3, p. 76
Leukoplakia buccalis, 192
Leydenia gemmipara, 130
Lips, 11, 79
Liver, Pl. 23, 25, 27, p. 25, 90
  abscess of, 208
  acute yellow atrophy of, 207
      prognosis of, 208
      treatment of, 208
  amyloid degeneration of, 209
  carcinoma of, 208
  cirrhosis of, Pl. 56, p. 207
    atrophic, 207
    complications of, 207
    first stage of, 207
    hypertrophic, 207
    second stage of, 207
    treatment of, 207
  constricted, Pl. 55, p. 209
  cyanotic, 209
    treatment of, 209
  diseases of, 209
    climatic treatment of, 236
    table of, 210
  echinococcus of, 208
    treatment of, 208
Liver, fatty degeneration of, 209
  melanosarcoma of the, Pl. 57
  nutmeg-, 209
  palpation of, 91
  percussion of, 92
  syphilis of, 208
    treatment of, 208
Localization of pain, 16
Low-pitched tympanitic note, 30
Ludwig's angina, 192
Lumbago, 228
Lung, Pl. 27
  abscess of, sputum from, Pl. 9
    staphylococci from, Pl. 10
  actinomycosis of, sputum from, Pl. 10
  gangrene of, sputum from, Pl. 9
  hemorrhagic infarction of, 175
  left, 41
  right, 40
Lungs, Pl. 23, 25, p. 14, 40
  anatomy of, 40
  auscultation of, 14, 46
  displacement downward of lower limits of, 43
    symptoms of, 44
    upward of lower limits of, 42
  dulness upon percussion of, 43
  inspection of, 42
  metallic note over, 45
  percussion of, 14, 42
  physiology of, 41
  spirometry of, 42
  topographic relations of, 40
  tympanitic note over, 44
  variations in percussion-note over, 45
  -tissue in sputum, 52
Lymphemia, 222

MAGNESIUM in urine, 103
  phosphate, basic, Pl. 15
Malaria, blood from a case of, Pl. 5
Malarial cachexia, 153
  fever, 151
    atypical varieties of, 153
    diagnosis of, 153
    incubation of, 152
    masked, 153
    onset of, 152
    pernicious, 153
    treatment of, 153
  plasmodia, Pl. 5
Manifestations, general, 6
Massage, 237

Meal, trial, 88
Measles (see Morbilli), 154
Measurement of abdomen, 15
Meat-poisoning, 232
  treatment of, 232
Medicaments, 237
  in urine, Pl. 21, p. 113
Medullary cells, Pl. 2
Megastoma entericum, 129
Melanin in urine, 113
  test for, Pl. 20
Melanosarcoma of the liver, Pl. 57
Membranous enteritis, 200
Meningitis, basilar, 136
  epidemic cerebrospinal, 149
      diagnosis of, 149
      sequelæ of, 149
      treatment of, 150
  tuberculous, 136
    diagnosis of, 137
    treatment of, 137
Mensuration of thorax, 13
Mercury, poisoning with, 229
    treatment of, 229
Metabolism, abnormalities of, examination of, 115
  bodily, disorders of, 224
  disorders of, climatic treatment of, 236
Metallic heart-sounds, 65
  note over lungs, 45
  percussion-note, 30
Metamorphosing breathing, 47
Meteorism, 94
Micrococci, 122
Micrococcus tetragenus, 123
Micro-organisms, pyogenic, Pl. 22, p. 26
Microscopy, 20
  instruments for, 20
Microsporon furfur, 121
Micturition, pain in, 18
Miliary tuberculosis, 136
Mirrors, examination with, 22
Mitral insufficiency, Pl. 45
  orifice, stenosis of, 57
  stenosis, compensated, Pl. 44
  valve, insufficiency of, 56
Molds, 120
Monocrotism, 70
Moore's (caramel-) test, Pl. 20
  test for sugar, 108
Morbilli, 154
  complications of, 154
  incubation of, 154
Morbilli, prodromes of, 154
  sequelæ of, 154
  treatment of, 154
Morphin, poisoning with, 231
    treatment of, 231
Mouth, 11, 79
  microscopy of, Pl. 7
Mucor corymbifer, 120
Mucus in stools, 97
Mumps, 157
Murexid test, 104
Murmurs, heart-, 33
  intestinal, 34
  systolic, over blood-vessels, 66
Musculature, 9
Mycosis, intestinal, 143
Myocarditis, 185
Myocardium, diseases of, 185
    treatment of, 185
  fatty degeneration of, 185
  fibroid degeneration of, 185

NARCOTICS, 241
Nasal catarrh, chronic, 161
  fetid, 162
  cavities, microscopy of, Pl. 7
  mucous membrane, secretion from, Pl. 7
Nasopharyngeal state, examination of, 35
Nasopharynx, adenoid vegetations of, 193
Neck, 12
Nematodes, 126
Nephritis, acute hemorrhagic, 211
      prognosis of, 212
      sediment from, Pl. 18
      symptoms of, 211
      treatment of, 212
      urine of, 212
  chronic, Pl. 18, p. 212
    diagnosis of, 213
    treatment of, 213
    parenchymatous, 212
    sediment from, Pl. 19
Nephrolithiasis, 216
  diagnosis of, 217
Neuralgia, 17
Neurotic disorders of the heart, 186
Neutrophile cells, 74
  granulation, Pl. 2
New-growths in the chest, 176
      symptoms of, 176
      treatment of, 177

Nicotin, poisoning with, 231
    treatment of, 231
Nitrates and nitrites in urine, 103
Nose, 11
  diseases of, 161
  examination of, 35
Nucleo-albumin, 107
Nutmeg-liver, 209
Nutrient enemata, 234
Nutritive state, 7
Nylander's test for sugar, 108

OBESITY, 226
  symptoms of, 226
  treatment of, 226
Occlusion of bowel, Pl. 64, p. 201
    diagnosis of, 201
    treatment of, 201
Occupation, 2
Oïdium albicans, 121
  lactis, 121
Oil of sandalwood in urine, 114
Oligochromemia, 75
Oligocythemia, 76
Orifices, cardiac, auscultation of, 14
Osteomalacia, 227
  prognosis of, 227
  treatment of, 227
Oxalic acid in urine, 105
Oxyures, 202
Oxyuris vermicularis, 126
Ozena, 162
  treatment of, 162

PACKS, wet, 235
Pain, 16
  character of, 16
  colicky, 17
  in evacuation of the bowels, 18
  in extremities, 18
  in face, 17
  in micturition, 18
  involving the trunk, 17
  localization of, 16
  pericarditic, 17
  peritonitic, 17
  pleuritic, 17
Palate, 12, 80
  paralysis of, 194
Palpation, 28
  of abdomen, 15
  of heart, 14
  of liver, 92
  of stomach, 84
Palpitation of heart, nervous, 186
Pancreas, diseases of, 211
    diagnosis of, 211
    treatment of, 211
Pancreatic cyst, Pl. 61
Paralysis of epiglottis, 40
  of posterior crico-arytenoid, 39
  of recurrent laryngeal nerve, 39
  of thyro-arytenoid muscle, 39
  of transverse arytenoid muscle, 39
  of vocal bands, 38
Parasites, 120
  animal, 126
  intestinal, 202
  of the sputum, Pl. 10
  vegetable, 120
Parotiditis, epidemic, 157
Patient, examination of, 1
Pediculi, 126
Peliosis rheumatica, 224
Pelvis of kidney, epithelial cells from, Pl. 16
Penicillium glaucum, 120
Peptone-test, Pl. 20
Percussion, 29
  clearness on, 30
  dulness on, 30
  of abdomen, 15
  of heart, 14, 62
  of kidneys, 99
  of liver, 92
  of lungs, 14, 42
  of stomach, 84
  tympanitic, 30
  -note, Biermer's change in, 45
    Friedreich's respiratory change in, 45
    Gerhardt's postural change in, 45
    metallic, 30
    over lungs, variations in, 45
    Wintrich's change in, 45
Percutory topography, Pl. 24
    of the back, Pl. 26
    of the lateral aspects of the trunk, Pl. 28
Perforative peritonitis, Pl. 67
Pericardial murmurs, 66
Pericarditic friction, 34
  pain, 17
Pericarditis, 186
  dry, 187
    prognosis of, 188
    symptoms of, 187
    treatment of, 188

Pericarditis, exudative, Pl. 49, p. 187
  fibrinous, 187
  hemorrhagic, 187
  tuberculous, 135
Pericardium, diseases of, 186
Peritoneum, carcinoma of, 205
  diseases of, 202
Peritonitic friction, 34
  pain, 17
Peritonitis, acute, 203
    symptoms of, 203
    treatment of, 204
  chronic, 204
    prognosis of, 204
    symptoms of, 204
  circumscribed, 203
  diffuse, 203
  perforative, Pl. 67
  purulent, 203
  putrid, 203
  serofibrinous, 203
  tuberculous, Pl. 66, p. 136
    treatment of, 136
Perityphlitis, Pl. 65, p. 202
  symptoms of, 203
  treatment of, 203
Pernicious anemia, Pl. 3
Personal, 1
Pertussis, 157
  catarrhal stage of, 157
  sequelæ of, 157
  spasmodic stage of, 157
  stage of decline of, 157
  treatment of, 157
Pharyngeal catarrh, chronic, 193
    treatment of, 193
  cough, 37
Pharyngitis, dry chronic, 193
Pharynx, 12, 79, 81
  paralysis of, 194
Phenols in urine, 105
Phenylhydrazin-test for sugar, 110
Phosphates, earthy, Pl. 15
  in urine, 103
Phosphatic calculi, 114
Phosphorus, poisoning with, 229
    treatment of, 229
Phthirius pubes, 126
Physical methods of treatment, 236
Pigment-cells, Pl. 8
Plague, 160
Plasmodia, 130
Plasmodial infections, 151
Pleura, puncture of, 49
Pleural cavity, exploratory puncture of, 14
  exudates, 49
  transudates, 50
Pleurisy, 172
  acute rheumatic, 146
    treatment of, 146
  diagnosis of, 167, 174
  dry, 172
  etiology of, 172
  exudative, Pl. 31, 32, p. 172
    on the right side, Pl. 33
  onset of, 172
  physical examination of, 172
  treatment of, 174
  tuberculous, 135
Pleuritic friction, 32
  pain, 17
Pneumococci from the blood, Pl. 5
  in pneumonic sputum, Pl. 10
  in sputum, 54
Pneumococcus, 122
Pneumonia, croupous, Pl. 29, 30, p. 147
    abnormal terminations of, 148
    complications of, 148
    diagnosis of, 148, 167
    prognosis of, 148
    treatment of, 149
  lobular, 168
Pneumokonioses, 171
Pneumopericardium, 189
  prognosis of, 189
Pneumotherapy, 237
Pneumothorax, Pl. 41, p. 175
  treatment of, 175
Poisoning, 228
Polarization-apparatus for detecting sugar, 109
  -method for estimating sugar, 111
Polyarthritis, acute rheumatic, 144
Portal vein, inflammation of, 209
    thrombosis of, 209
Posture, 7
Potassium in urine, 103
Precordium, inspection and palpation of, 61
  thrill in, 62
  visible pulsation in, 61
Preparations, stained, 21
  unstained, 21
Present state, 6
Projection of internal organs upon the anterior aspect of the body, Pl. 23

Projection of internal organs upon the lateral aspects of the trunk, Pl. 27
    upon the posterior aspect of the body, Pl. 25
Proteus vulgaris, Pl. 22
Protozoa, 129
Pseudoleukemia, 223
Puberty, diseases of, 1
Pulex irritans, 126
  penetrans, 126
Pulmonary abscess, 171
  emphysema, Pl. 38
  gangrene, 170
  infarction, Pl. 37
  tuberculosis, 132
    incipient, Pl. 39
    progressive, Pl. 40
Pulsation, 13
  visible, in the precordium, 61
Pulse, 11, 14, 67
  acceleration of, 67
  celerity of, 68
  hardness of, 68
  retardation of, 67
  rhythm of, 68
  size of, 68
Puncture, exploratory, 23
    of abdomen, 15
    of pleural cavity, 14
  of pleura, 49
Purpura, 224
Pus from gonorrhea, Pl. 19
  from tuberculous cystitis, Pl. 19
  in stools, 98
  -cells, Pl. 16
Pustule, malignant, 143
Pyelitis, calculous, sediment from, Pl. 18
Pyelonephritis, 215
  diagnosis of, 216
  symptoms of, 216
  treatment of, 216
Pyemia, blood from, Pl. 5
Pyloric stenosis, 199
Pylorus, cicatricial stenosis of the, Pl. 52
  dilatation of the stomach, with carcinoma of the, Pl. 53
Pyogenic micro-organisms, Pl. 22, p. 26
Pyopneumothorax, Pl. 42

RACHITIS, 227
  physical signs of, 227
Rachitis, treatment of, 227
Râles, 32, 47
  crepitant, 47
  dry, 47
  metallic, 48
  moist, 47
  ringing, 48
Recurrent laryngeal nerve, paralysis of, 39
Redness of vocal bands, 38
Reduction-cure, 234
  -tests for glucose, 107
Relapsing fever, 151
    diagnosis of, 151
    incubation of, 151
    spirilla of, Pl. 5, p. 125
    treatment of, 151
Remittent fever, 9
Renal epithelium, Pl. 16
Respiration, 13
  type of, 13
Respiratory apparatus, 35
    examination of, 35
  muscles, 12
  organs, diseases of, 161
      climatic treatment of, 236
Rest-cure, 233
Retraction, systolic, 61
Retropharyngeal abscess, 81, 193
Rheumatic diseases, 144
  torticollis, 228
Rheumatism, muscular, 228
    treatment of, 228
Rhinitis, acute, 161
Rhinoscopy, anterior, 35
  posterior, 36
Rhythm, galloping, 65
Roborants, 241
Rötheln (see Rubeola), 155
Round worms, 126

SALICYLIC acid in urine, Pl. 21
Salkowski's test for albumoses, 107
Sandalwood, oil of, in urine, 114
Sarcina ventriculi, 123
Sarcoma of the kidney, Pl. 68
Sarcoptes scabiei, 126
Sausage-poisoning, 232
  treatment of, 232
Scarlet fever, 155
    complications of, 155
    incubation of, 155
    prodromes of, 155
    sequelæ of, 155
    treatment of, 155

Scorbutus, 223
  treatment of, 224
Season, 3
Sediments, 22
  crystalline urinary, Pl. 14, 15
  urinary, Pl. 13
    attending diseases of bladder and kidneys, Pl. 18
    organized, Pl. 16
Septicopyemia, cryptogenetic, 146
    diagnosis of, 147
    prognosis and treatment of, 147
Serum-test for typhoid fever, 140
Sex, 2
Shape of thorax, 13
Skeleton, 7
Skin, 7
Sleep, 5
Small-pox (see Variola), 155
Social relations, 2
Sodium in urine, 103
  urate in urine, Pl. 13
Special condition, 11
Spectra, normal solar, Pl. 6
Spectroscopy of the blood, 74
Spermatozoa, Pl. 16
Sphygmography, 69
Spirilla of relapsing fever, Pl. 5, p. 125
Spirillum-infections, 151
Spirochæta buccalis, 125
Spirochetæ from gangrenous stomatitis, Pl. 7
Spirometry of lungs, 42
Splashing-sounds, 34
Spleen, Pl. 25, 27, p. 92
  diseases of, 210
  displacement of, 93
  enlargement of, 93, 210
    treatment of, 211
  palpation of, 93
  wandering, 211
Splenic dulness, abolition of, 93
Splenomedullary leukemia, Pl. 2, 60, p. 222
Spool-worm, 126
Spotted fever, 159
Sputum, 15, 50
  actinomyces-granules in, 54
  anthrax-bacilli in, 54
  aspergillus-mycelium in, 54
  bacteria in, 53
  cardiac-lesion cells in, 52
  cellular elements in, 52
  Charcot-Leyden crystals in, 53
Sputum, cholesterin-plates in, 53
  color of, 51
  crystals in, 53
  Curschmann's spirals in, 53
  Dittrich's plugs in, 53
  echinococcus-hooklets in, 53
  elastic fibers in, 53
  eosinophile cells in, 52
  fibrinous coagula in, 51
  from a case of tuberculosis, Pl. 10
  from abscess of lung, Pl. 9
  from actinomycosis of the lung, Pl. 10
  from gangrene of lung, Pl. 9
  from influenza-bronchitis, Pl. 10
  hematoidin-crystals in, 53
  in layers, 51
  influenza-bacilli in, 54
  leucin-spheres in, 53
  leukocytes in, 52
  lung-tissue in, 52
  microscopy of, Pl. 8, p. 52
  parasites of, Pl. 10
  pneumococci in, 54
    pneumonic, Pl. 10
  thrush-fungus in, 54
  tissue-elements in, 52
  tubercle-bacilli in, 53
  tyrosin-needles in, 53
Stained preparations, 21
Staining, 24
  solution, 78
Staphylococci from abscess of lung, Pl. 10
Staphylococcus pyogenes, 122
    albus, Pl. 22
State, present, 6
Stenocardia, 186
Stenosis, cicatricial, of the pylorus, Pl. 52
  mitral, compensated, Pl. 44
  of aortic orifice, 59
  of bowel, 201
  of esophagus, carcinoma of the cardia, with, Pl. 51
  of mitral orifice, 57
Stomach, Pl. 23, 27, p. 82
  anatomy of, 82
  carcinoma of, Pl. 54, p. 197
    symptoms of, 197
    treatment of, 197
  diet for diseases of, 233
  dilatation of, 198
    prognosis of, 198
    symptoms of, 198

Stomach, dilatation of, treatment of, 198
  with carcinoma of the pylorus, Pl. 53
 diseases of, 195
  climatic treatment of, 236
 inspection of, 84
 microscopy of, Pl. 11
 normal and pathologic position of, Pl. 51
 palpation of, 84
 percussion of, 84
 physiology of, 83
Stomachics, 239
Stomatitis, 190
 aphthous, 191
 catarrhal, 190
 gangrenous, spirochetæ from, Pl. 7
 necrotic, 191
 treatment of, 191
 ulcerative, 191
Stools, 97
 blood in, 97
 calculi in, 98
 color of, 97
 mucus in, 97
 pus in, 98
Streptococcus pyogenes, Pl. 22, p. 122
Subcutaneous connective tissue, 7
Subfebrile temperature, 9
Subjective symptoms, 16
Succussion, Hippocratic, 48
 -sounds, 33
Sucking worms, 129
Sugar, quantitative determination of, 110
 Trommer's test for, Pl. 20
Sulphates in urine, 102
Swallowing sound, 34
Sweating, 5
 abnormal, 8
Swelling of vocal bands, 38
Symmetry of abdomen, 15
 of thorax, 13
Symptoms, subjective, 16
Syphilis of bowel, 200
  symptoms of, 201
  treatment of, 201
 of larynx, 164
 of liver, 208

TÆNIA echinococcus, 128
 nana, 128
Tænia saginata, 128
 solium, 128
Tape-worm, 202
 treatment of, 202
Tape-worms, 128
Teeth, 11, 79
Temperature, bodily, 9
 hyperpyretic, 9
 subfebrile, 9
Tetanus-bacilli, 124
Therapeutic notes, 233
Thermometry, 23
Thirst, 5
Thorax, 13
 mensuration of, 13
 shape of, 13
 symmetry of, 13
Thrill in the precordium, 62
Throat, 5
Thrush-deposits, 191
 -fungus in sputum, 54
Thyro-arytenoid muscle, paralysis of, 39
Thyroid gland, 12
Tissue-elements in sputum, 52
Toadstool-poisoning, 231
 treatment of, 231
Tongue, 11, 80
 geographic, 192
Tonsillitis, 192
Tonsils, 12, 80
 chronic hypertrophy of, 193
  treatment of, 193
Topical applications, 238
Topography, percutory and auscultatory, Pl. 24
  of the back, Pl. 26
  of the lateral aspects of the trunk, Pl. 28
Torticollis, rheumatic, 228
Trachea, examination of, 36
Transmission of voice, 32
Transudates, pleural, 50
Trematodes, 129
Trichina spiralis, 127
Trichinosis, 160
 diagnosis of, 161
 treatment of, 161
Trichocephalus dispar, 126
Tricophyton tonsurans, 121
Tricuspid insufficiency, Pl. 47
 valve, insufficiency of, 59
Trommer's test for sugar, Pl. 20, p. 108
Trunk, pain involving, 17

Trunk, percutory topography of lateral aspects of, Pl. 28
  projection of internal organs upon lateral aspects of, Pl. 27
Tube-casts, urinary, Pl. 17
Tubercle-bacilli, 131
    in sputum, 53
  -bacillus, 123
Tuberculosis, generalized, 136
  glandular, 134
    treatment of, 134
  intestinal, 134, 200
    treatment of, 135
  laryngeal, 134
    complications of, 134
    treatment of, 134
  localized, 132
  miliary, 136
  of kidney, 217
  pulmonary, 132
    advanced, symptoms of, 132
    climatic treatment of, 236
    complications of, 133
    first symptoms of, 132
    incipient, Pl. 39
    progressive, Pl. 40
    terminal symptoms of, 132
    treatment of, 133
  sputum from, Pl. 10
  urogenital, 135
    treatment of, 135
Tuberculous cystitis, 219
    pus from, Pl. 19
  diseases, 131
  meningitis, 136
  pericarditis, 135
  peritonitis, Pl. 66, p. 136
  pleurisy, 135
Tumors of vocal bands, 38
Tylosis, 192
Tympanitic note over the lungs, 44
    high-pitched, 30
    low-pitched, 30
  percussion, 30
Type of respiration, 13
Typhoid fever, 137
    blood-preparation from a case of, Pl. 3
    complications of, 139
    convalescence from, 139
    diagnosis of, 139
    incubation of, 138
    initial stage of, 138
    prodromes of, 138
    prognosis of, 140
Typhoid fever, relapses of, 139
    second stage of, 138
    serum-test for, 140
    third stage of, 139
    treatment of, 140
  -bacilli, Pl. 22, p. 27, 124, 137
Typhus fever, 159
    incubation of, 159
    prodromal period of, 159
    treatment of, 159
Tyrosin, Pl. 15
  -needles in sputum, 53

ULCER, duodenal, 197
  gastric, 196
Ulceration of vocal bands, 38
Undefined breathing, 47
Unstained preparations, 21
Urea in urine, 104
Urethra, squamous epithelium from, Pl. 16
Uric acid from a gouty nodule, Pl. 21
    in urine, 104
Uric-acid crystals in urine, Pl. 13
  diathesis, 225
    etiology of, 226
  sediment in urine, Pl. 13
Urinary sediments, Pl. 13
    attending diseases of the bladder and the kidneys, Pl. 18
    crystalline, Pl. 14, 15
    organized, Pl. 16
  tube-casts, Pl. 17
Urine, 16, 100
  acetone in, 112
  albumin in, 105
  ammonia in, 103
  amount of, 100
  antifebrin in, 113
  antipyrin in, Pl. 21, p. 113
  balsam of copaipa in, 114
  biliary acids in, 112
    coloring-matter in, 112
  blood in, 111
  bromin in, 114
  calcium in, 103
  carbolic acid in, 114
  carbonates in, 103
  chemic examination of, 102
  chlorids in, 102
  color of, 101
    -reactions of, Pl. 20
  creatinin in, 105
  diacetic acid in, 112

Urine, fat in, 113
  hippuric acid in, 105
  hydrogen sulphid in, 113
  hypoxanthin in, 105
  indican in, 104
  iodin in, 114
  lead in, 113
  magnesium in, 103
  medicaments in, Pl. 21
  melanin in, 113
  nitrates and nitrites in, 103
  normal inorganic constituents of, 102
    organic constituents of, 104
  oil of sandalwood in, 114
  oxalic acid in, 105
  pathologic ingredients of, 105
  phenols in, 105
  phosphates in, 103
  potassium in, 103
  reaction of, 101
  rhubarb in, 114
  salicylic acid in, 114
  santonin in, 114
  sediment in, 101
  senna in, 114
  sodium in, 103
    urate in, Pl. 13
  specific gravity of, 101
  sulphates in, 102
  turpentine in, 114
  urea in, 104
  uric acid in, 104
    crystals in, Pl. 13
  xanthin in, 105
  yellowish friable sediment in, Pl. 13
Urogenital tuberculosis, 135
Uropoietic organs, diseases of, 211
  system, examination of, 99

VALVULAR lesions of the heart, 180
    theory of, 55
Varicella, 157
Variola, 155
  complications of, 157
  eruptive stage of, 155
Variola, incubation of, 155
  modifications of, 156
  prodromes of, 155
  sequelæ of, 157
  stage of desiccation of, 156
  stage of invasion of, 155
  suppurative stage of, 155
  treatment of, 157
Varioloid, 156
Veins, examination of, 67
Venous hum, 34, 67
  pulse, systolic, 67
Ventricle, left, hypertrophy of, 184
  right, hypertrophy of, 185
Vermes, 126
Vesicular breathing, enfeebled, 46
    exaggerated, 46
    pathologic, 46
  respiratory murmur, 31
Vessels, 14
  diseases of, 189
Vigor, bodily, 4, 7
Vocal bands, paralysis of, 38
    redness of, 38
    swelling of, 38
    tumors of, 38
    ulceration of, 38
Voice, auscultation of, 48
  transmission of, 32
Vomited matter, constitution of, 89
Vomiting, 88

WAXY tube-casts, Pl. 17
Weil's disease, 206
Werlhof's disease, 224
Whooping-cough (see Pertussis), 157
Wintrich's change in percussion-note, 45

XANTHIN in urine, 105
  -calculi, 114

YEAST-FUNGI, 121
Yellow fever, 160
    treatment of, 160
Yellowish friable sediment in urine, Pl. 13

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See page 18 for a List of Contents classified according to subjects

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